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BJÖRKMAN (E.). **Über die Bedingungen der Mykorrhizabildung bei Kiefer und Fichte.** [On the conditions of mycorrhiza formation in Pine and Spruce.]—*Symb. bot. upsaliens.*, vi, 2, 190 pp., 34 figs., 28 graphs, 1942. [English summary. Received August, 1945.]

The writer's field studies failed to yield any clue to the wide and baffling discrepancies in mycorrhizal development on pine and spruce in Sweden [*R.A.M.*, xxii, p. 266]. The results obtained in soil culture experiments indicated that light is the factor of paramount importance in mycorrhizal formation, which was favoured by an increase of illumination up to the maximum value tested, i.e., half that available in the open. No mycorrhiza developed at $\frac{1}{16}$ or even, in soil with high nitrogen content, at $\frac{1}{8}$ of full light. Extreme nitrogen or phosphorus deficiency was found to impede mycorrhizal growth, but a moderate shortage of one or other of these elements is a condition of infection [ibid., xvii, p. 126]. When the available nitrogen content was not too high, mycorrhizal infection was favoured by all additions of phosphorus tested. In aseptic flask cultures of pine seedlings inoculated with *Rhizopogon roseolus* or *Boletus variegatus*, moderate additions of glucose promoted mycorrhizal invasion, notably in weak light. The roots of seedlings grown under conditions conducive to mycorrhizal development contained larger amounts of reducing substances and reacted much more intensively to the potassium iodide starch test than those deprived of the factors necessary for infection.

On the basis of these data it is concluded that the mycorrhizal fungi enter the host roots in search of soluble carbohydrates [ibid., xix, p. 37], which will only accumulate in sufficient quantities where the moderate scarcity of an essential nutrient prevents rapid photosynthesis. This theory would account for the complex interrelationships existing between mycorrhizal infection, light, and available nitrogen and phosphorus, and explain all or most of the diversities of mycorrhizal habit observed among conifers, both under natural and controlled conditions. It would further appear that nodule development in alders [cf. ibid., xxi, p. 34] and legumes is influenced by a similar interplay of environmental factors.

GÄUMANN (E.) & JAAG (O.). **Über induzierte Abwehrreaktionen bei Pflanzen.** [On induced defence reactions in plants.]—Reprinted from *Experientia*, i, 1, 3 pp., 1 fig., 1945.

The roots of *Loroglossum* (*Himantoglossum*) *hircinum*, an orchid growing on rocky heathland in the Jura Mountains, Switzerland, are infected by a mycorrhizal fungus, *Rhizoctonia repens* [*R.A.M.*, xxii, p. 171], which does not normally occur in the rhizomes. When the fungus is grown on a suitable medium in proximity to a fair-sized piece aseptically excised from a rhizome, the former is halted in its advance towards the latter, evidently by the diffusion of an anti-fungal substance. Rhizomes killed by chloroform narcosis or cold prior to testing failed to react defensively to the fungus, which completely invested them. This observation is interpreted as indicating that the anti-fungal substance originates as a true

defensive reaction to the advancing metabolic products of the invader and is not present as a natural ingredient of the rhizome from the outset.

Such defensive reactions are not confined to the rare *L. hircinum* but occur also in common species, such as *Orchis militaris* and *O. morio*, the rhizomes of which are similarly free from fungal infestation.

FERNOW (K. H.). **Relation of sample size to accuracy.**—*Amer. Potato J.*, xxi, 8, pp. 229–234, 1944.

In this paper the author discusses the sampling technique of seed-potato inspectors and tabulates the probability of obtaining a given number of diseased plants in a sample of given size from a stock containing a given percentage of disease, and the minimum and maximum percentages of diseased tubers likely to be found in samples of varying size from stocks containing given percentages of disease.

MORGAN (E. T.). **The use of the tuber-unit plots in the production of seed Potatoes.**—*J. Dep. Agric. W. Aust.*, xxii, 1, pp. 70–74, 2 figs., 1945.

The tuber-unit system was introduced by the author in 1937 with a view to improving the system of roguing potatoes, subject to Government inspection under the Certified Seed Potato Scheme. Under the unit system, tubers, sufficiently large to allow four or more seed pieces to be cut from one potato, are cut into sets, those from each tuber, comprising a unit, being planted consecutively about 15 in. apart along the row. A double spacing of 30 in. should be left between each succeeding unit. Plants should be carefully inspected during growth and should any seed piece of the unit produce a diseased plant, the whole unit is discarded.

Tuber-unit seed-potato plots have the following advantages: (1) greater certainty of detecting virus-diseased plants by reason of a number of such plants being together; (2) earlier detection of such diseases, allowing roguing to be undertaken sooner and thereby lessening the risk of transmission; (3) limiting the points of infection for the transmission of the disease.

HUNTER (J. G.) & M'GREGOR (A. J.). **An investigation of diseases resulting from inadequate nutrition of the Potato plant.**—*Scot. J. Agric.*, xxv, 3, pp. 176–178, 1945.

Notes are given on three types of disease observed on potatoes in Scotland. In the first, the plants were dwarfed, and the young leaves had a slight upward roll, which became more marked as the plants grew older. In August, when the haulms were about 6 in. high, the older leaves showed yellow and brown blotches and dead patches. This marking was most apparent at the margins and tips, though in the oldest leaves it was also present between the veins. The affected leaves died prematurely and fell off. The younger leaves were a pale, unhealthy green. The tubers were small and the yield was reduced. Analyses of diseased plants and of the soil indicated that the condition was primarily due to a serious calcium deficiency in the soil, and was aggravated by low magnesium. Treatment of the highly acid soil with ground shell lime greatly increased the yield.

In the second type of disease, growth was very poor, in spite of adequate supplies of potato manure. The haulms in August were only 12 to 18 in. high and very bare, as a result of the premature death and shedding of many of the older leaves. In the early stages, the leaf margins had a slight upward curl. Yellowing of the margins and tips soon set in, and changed to an orange-yellow colour which extended from the margins to the veins and midribs. In some cases, the only green parts left on a leaf were small patches between the veins. Brownish-purple areas were generally present between the veins, sometimes surrounded by an almost normal green colour, and at others by yellow tissue. Brown, dead patches were

often present on the leaves. The yield was considerably reduced. The affected leaf blades contained only one-seventh as much magnesium as did healthy ones, and the disease is attributed to magnesium deficiency. This soil was also very acid. The best method of preventing magnesium deficiency is to apply lime (particularly dolomitic limestone) or to use manure; alternatively suitable dressings of magnesium salts can be applied.

In the third type of disease, the plants were stunted, and parts of the leaves were yellow-green, though no marginal effect was noted. The extreme edges of the leaves were brownish, with a slight upward curl. Tuber yields were reduced. Affected plants had an abnormally low nitrate content. Control consists in applying nitrogenous manures.

SANFORD (G. B.). **Common scab of Potato in dry and wet soils.**—*Sci. Agric.*, xxv, 9, pp. 533–536, 1 fig., 1945.

An experiment is described in which batches of soil, containing water to approximately 19, 22, 26, 30, 35, and 39 per cent. of their water-holding capacity, were steam-sterilized and aseptically poured into sterilized metal containers with holes and glass windows in their covers. These were about half filled, and a narrow band of soil next to the window was seeded with a small quantity of a soil-spore mixture obtained by shaking a little soil over a matured culture of *Actinomyces scabies*. The containers were then filled up, the holes plugged with cotton, and the edges of the glass windows sealed with paraffin wax. The incubation temperature was 23° C. The first soil of this series was too dry for plant growth and the last one too wet to handle. Comparative ratings of the vegetative growth of *A. scabies* were made on the fifth, sixth, and ninth days after seeding with spores, a value of 10 representing the best growth on each date.

Five days after seeding, growth in the dry soil (19 to 26 per cent. moisture) was 30 per cent. poorer than that in the soil of optimum or over-optimum moisture (30 and 35 per cent. moisture, respectively), but about 70 per cent. better than in the wet soil (39 per cent. moisture). One day later, growth in the dry soil was only 25 per cent. better, and after three days the situation was reversed, with growth 12 per cent. better in the wet soil. The fungus appears to grow surprisingly well in a soil too dry for plant growth. It is concluded that in the absence of effective antagonism from associated saprophytes, severe scab may occur in soils high in moisture content, as well as in drier soils.

GÄUMANN (E.) & HÄFLIGER (E.). **Der Einfluss der Bodentemperatur auf die Entwicklung und den Schorfbefall der Kartoffelknollen.** [The influence of the soil temperature on the development and scab infection of Potato tubers.]—Reprinted from *Phytopath. Z.*, xv, 1, 23 pp., 1 fig., 16 graphs, [undated, received September, 1945].

Studies on the influence of the soil temperature on potato tuber development and scab (*Actinomyces scabies*) infection have been carried out by various workers and summarized, e.g., by Wollenweber (in Sorauer's 'Handbuch der Pflanzenkrankheiten', Part II, Fifth edition [*R.A.M.*, xi, p. 527]) and Roemer *et al.* [ibid., xviii, p. 196]. According to these authorities, the optimum for tuber infection lies between 25° and 28° C., while the pathogen ceases to grow at 8° though developing freely up to 38°. Field observations in Switzerland, however, gave rise to doubts as to the universal applicability of these data and suggested the possibility of the intervention of other physiologic races of the fungus in mountainous countries. Experiments were therefore conducted by Gäumann and Riethmann's method (*Phytopath. Z.*, xi, pp. 617–631, 1938) to determine the effect of soil temperatures on host and parasite, using Swiss mountain material of the early variety Bintje in 1942 and of the medium-late Ideal in 1943.

The optimum soil temperature for tuber yield was found to lie within the broad range of 13° to 26°. Inside this range both the number of tubers per plant and their average weight reached a maximum. The influence of soil temperature was noticeable in the periderm, and especially the primary cortex, which (except for the outermost layer) constitutes a reservoir for starch. Below 27° the starch-free zone measures less than 100 μ in thickness, above 30° it rapidly rises to 300 μ, and at the upper limit of 34° the entire tuber is practically devoid of starch. The contents both of glucose and starch attain a well-marked optimum at 20° to 22° and then sink rapidly, especially at higher temperatures.

The minimum, optimum, and maximum temperatures for the growth of the strain of *A. scabies* used in the trials were 3°, 24°, and 33°, but scab infection assumed its maximum extent and virulence at a suboptimum for the pathogen (10° in the case of Bintje and 13° in that of Ideal). These temperatures coincided with the greatest productivity of the tubers, indicating the preference of the parasite for plants with a high degree of vitality.

Taylor (G. G.). **Trials with Bordeaux mixture, copper oxychloride, and copper oxide sprays for control of late-blight (Phytopthora infestans) of Potatoes.**—*N.Z. J. Sci. Tech.*, *A*, xxvii, 1, pp. 4–8, 1 fig., 1945.

Spraying with Bordeaux mixture 3–4–50 and the copper oxychlorides, cuprox and oxicop (5 lb. in 100 gals.), effectively combated late blight (*Phytophthora infestans*) in King Edward potatoes in the Auckland district of New Zealand from 1940 to 1943. No advantage was derived from the use of a higher concentration (5–4–50) of Bordeaux, or from the admixture of cottonseed oil with the fungicide. There was some evidence that perenox (5–100) caused yield reductions through spray damage effects.

Melhus (I. E.). **Late blight forecasting service.**—*Phytopathology*, xxxv, 6, pp. 463–479, 1 graph, 1945.

In 1943, potato late blight [*Phytophthora infestans*] forecasting [*R.A.M.*, xvi, pp. 514, 832; xvii, p. 59, *et passim*] was undertaken by a subcommittee of the 'War Service Committee of the Upper Mississippi Valley' Plant Pathologists in consequence of an estimated loss of 25,000,000 bushels in the previous year, which demanded prompt co-operative action on the part of all States and the two Prairie Provinces of Canada lying within the region. Temperature and rainfall records from 1941 to 1944, inclusive, are presented for nine States, while in Missouri, Kansas, and Saskatchewan the incidence of infection was too low to justify any attempt at the correlation of meteorological data and the development of the pathogen. However, even allowing for incompleteness and possible errors in the records, it is significant that the destructiveness of late blight outbreaks can be predicted by a study of the influence of temperature and precipitation on the development of the causal organism.

During the period under review, a temperature of 70° F. represented the maximum conducive to luxuriant initial growth of the fungus, and in no case did the disease assume a virulent character where the mean temperature in June and July exceeded this figure, which was therefore of great assistance in the formulation of a basis for predictions as the reports arrived from committee members. The forecasting was based upon weekly weather reports, and in general this basis was satisfactory for forecasts of the prevalence and severity of *P. infestans* throughout the growing seasons of the four years covered by the survey.

In comparison with 1941 and 1942, 1943 and 1944 were marked by a noteworthy increase in control practices. In North Dakota, for instance, the number of spraying and dusting machines rose from 600 to 1,200 during the period of the investigations, and a similar upward trend was observed in other States. In

Minnesota six aeroplane dusters were operating in 1944. These vigorous efforts at combating the pathogen were doubtless in part responsible for its diminishing intensity of attack in the two later years.

JOHANSEN (GUDRUN). **Indsunkne Smaapletter paa Kartoffelknolde.** [Small, sunken spots on Potato tubers.]—*Tidsskr. Planteavl*, xlvii, pp. 675–682, 1943. [Received August, 1945.]

Of recent years, potato tubers in Denmark have been observed bearing hard, dry spots, ranging from pale through dark brown to greyish-black and sharply delimited from the healthy portion by a narrow, dark brown, moist zone consisting of suberized tissue. The trouble, which threatens to assume a virulent form, is usually only discovered in the spring, when the tubers are brought in from the clamps, and even then the symptoms may not appear until several weeks have elapsed. Although it is considered highly probable that the disease is caused by *Alternaria solani* [see next abstract] the fungus had not, at the time of writing, actually been isolated from infected material, very likely because of the simultaneous presence in the tissues, at this advanced stage of decay, of numerous secondary organisms, including *A. tenuis*, which overrun the original pathogen.

Important contributions to the study of early blight in other countries are summarized, with special attention to those of Klaus in Germany [*R.A.M.*, xx, p. 417].

STAPEL (C.). **Kartoflens Bladpletsyge.** [Potato leaf spot disease.]—Reprinted from *Ugeskr. Landm., 1943*, 29, 2 pp., 1943.

Potato leaf spot [early blight: *Alternaria solani*], which has assumed increasing importance of recent years in Denmark [see preceding abstract], was effectively combated on King Edwards at the State Phytopathological Experiment Station in 1940 by two applications of 2 per cent. Bordeaux mixture, the first on 23rd July and the second on 16th August, at a dosage of 1,600 l. per ha., the incidence of infection being reduced from 91·3 to 23·1 per cent. and the tuber yield increased from 518·7 to 538·5 hectokg. per ha. In another test on the Majestic variety the same procedure was followed, except that the fungicide was used at a dosage of 2,000 l. per ha., with a resultant drop in the amount of early blight from 91 to 15·3 per cent. and a corresponding increase in yield from 440·4 to 472·6 hectokg. per ha. In 1941 Bordeaux mixture (2 per cent.) was again applied to Majestics on 19th July and 18th August at 1,200 l. per ha., effecting a reduction in the incidence of *A. solani* from 100 to 64·3 per cent. and raising the yield from 308·1 to 347·5 hectokg. per ha. In this case the attack commenced early, and by 5th August two-thirds of the leaves were already infected. The 13 per cent. improvement in yield is attributed largely to the control of a late but virulent outbreak of blight [*Phytophthora infestans*], which was negligible in 1940, when the increase in production from spraying was proportionately small.

HOUSTON (B. R.). **Culture types and pathogenicity of isolates of Corticium solani.**—*Phytopathology*, xxxv, 6, pp. 371–393, 5 figs., 1 graph, 1945.

On the basis of cultural characters and observed pathogenicity, 52 isolates of *Corticium solani* were selected from 260 collections on 15 Californian crop plants for further pathogenicity studies [*R.A.M.*, xx, p. 391; xxiv, p. 286, *et passim*]. They were divided into three culture types, of which (A) was characterized by a heavy, stroma-like layer on the surface of the medium, nearly white at first, turning pale drab-grey to light drab (Ridgway), often convoluted; natal-brown sclerotia, frequently radiating out from the point of inoculation; a sparse, white to cinnamon aerial mycelium; a rapid growth rate (1 to 1·8 mm. per hour); and commonly a distinct darkening of the medium; (B) by no well defined, superficial, stromatic layer; few, natal-brown, globular sclerotia, 1 to 4 mm. in diameter; very profuse,

pale olive to sayal-brown mycelium; a moderate growth rate (0·6 to 0·8 mm. per hour); and no darkening of the substratum; (C) by a slight, vinaceous-buff, later wood-brown, superficial stromatic layer; natal-brown, globular sclerotia with a very irregular surface, often united into groups up to 15 mm. in diameter, while the scattered individuals measured 1 to 5 mm.; little or no aerial mycelium; a moderate to slow growth rate (0·5 to 0·7 mm. per hour); and moderate or no darkening of the medium. The hosts of type (A) comprised beans (pink (*Phaseolus vulgaris*), Lima [*P. lunatus*], and Mung [*P. mungo*]) [ibid., xxiv, pp. 88, 261], cotton, Blackeye cow-peas, fenugreek [*Trigonella foenum-graecum*], potato, rhubarb, spinach, vegetable marrow, sugar beet, and tomato; (B) was isolated exclusively from dry-rot cankers on sugar beet [ibid., xx, p. 391]; and (C) was collected on lucerne, asparagus, pink beans, celery, potato, strawberry, and tomato.

The results of tests to determine the effects of temperature on 25 isolates agreed in the main with those of other workers on *C. solani*. The optimum in the majority of cases was 28° C. and the maximum 42°, but for the type (C) isolates the corresponding figures were 25° and 33°, respectively, a differential feature placing the representatives of this group in a separate temperature category.

The pathogenicity of 26 isolates of type (A), 9 of (B), and 13 of (C) was tested on lucerne, cotton, spinach, sugar beet, and tomato, and the following average percentages of infection were secured with the three groups: (A) 82, 79, 64, 73, and 53, respectively; (B) 10, 4, 1, 7, and 3; and (C) 7, 5, 5, 7, and 2. In another series on bean the average percentages of diseased seedlings inoculated with the same isolates of (A), (B), and (C) were 86, 17, and 5, respectively. Thirteen isolates of (A), five of (B), and eight of (C) were also tested for their action on White Rose potato tubers, which gave rise to 42, 2, and 13 per cent. diseased shoots, respectively. Sclerotial production was abundant only on the stems and tubers infected with the (C) isolates, of which potato is the most common natural host, though the effects of (A) were more destructive.

Inoculation tests with 26 isolates of *C. solani*, of which 11, 6, and 9 fell into the (A), (B), and (C) groups, respectively, on sugar beet (*a*) roots and (*b*) crowns resulted in the following average infection percentages: (A) 85 and 65 on (*a*) and (*b*), respectively; (B) 97 and 17; and (C) 19 and 4. Members of group (B) were only able to enter through the enlarged roots, unlike the majority of (A) isolates, which also attacked the petioles and crown tissues. The relatively low incidence of disease induced by most of the (C) strains, which in nature are primarily associated with potato black scurf, marks them off as a distinct group in respect of pathogenicity to sugar beet, as already shown by Le Clerg [loc. cit.].

On the other hand, one of the (A) isolates from potato caused severe injury to sugar beet (100 and 93 per cent. on roots and crowns, respectively).

It is apparent from these observations that the cultural characters of *C. solani* isolates are more important in the prediction of their potential injuriousness to specific hosts than the source of their derivation.

Garrard (E. H.). **A storage rot of Potatoes caused by a fluorescent organism resembling Pseudomonas fluorescens (Flügge) Migula.**—*Canad. J. Res.*, Sect. C, xxiii, 3, pp. 79–84, 5 figs., 1945.

The author describes the isolation from potatoes, dug on allotments near Toronto, believed to have been stored satisfactorily and found rot-infected when sliced for cooking, of an organism considered to belong to a subgroup [*R.A.M.*, xiv, p. 16] of *Pseudomonas fluorescens*, consisting of non-plant pathogens and some weak parasites, and not unlike Lacey's strain 221 [ibid., xi, p. 700], isolated from potato tuber ring disease, and *Bacterium xanthochlorum* [*P. xanthochlora*; ibid., v, p. 574].

On examination the allotment tubers were found to be flabby; some bore no outward signs of infection, others had surface discoloration or showed scab, but

no cracks. A hard, black rot, a corky vascular decay, and brown to black areas with cracks and cavities in some cases, radiating towards the outside, were disclosed on cutting up the potatoes. When freshly cut, the tubers were hard, with little exudate, no ropiness, and no pronounced smell. Rotting at room temperature was not nearly so rapid as when kept at 5° C.

Although the symptoms resembled those of *Corynebacterium sepedonicum* [ibid., xxiv, p. 201], that organism was not found on Burkholder's medium [ibid., ix, p. 695], but plates supporting nutrient agar produced flat, greyish-white, circular to lobate colonies of non-sporing, Gram-negative, aerobic rods, measuring 1·5 to 3 by 0·7 to 0·9 μ, motile by one to three polar flagella [ibid., v, p. 574], and grouped singly or in pairs. The surrounding medium was distinguished by a glistening, yellowish-green fluorescence.

Inoculation of the isolated organism on sterile potato slices produced extensive black rot in 24 hours at 5°, with a slighter effect at 18° and 25°. The disease also developed in sound, whole tubers inoculated through a stab made by a sterile needle. After successive transfers on culture media, the organism lost its pathogenicity within six months. This fact and its virtual identity with *P. fluorescens* in culture have restrained the author from classifying the organism as a separate plant pathogen. It is suggested that the potatoes forming the subject of this enquiry were impaired for resistance to the organism by storage under home conditions.

Crépin (C.) & Arnaud (G.). **Les maladies de la Pomme de terre.** [Potato diseases.] —67 pp., 18 col. pl., 7 figs., Paris, Ministère de l'Agriculture et du Ravitaillement, 1943. [Received September, 1945.]

In this publication, based on earlier ones by Ducomet and Foëx [*R.A.M.*, iii, p. 98], and Ducomet, Foëx, and Alabouvette [ibid., xiv, p. 786], the authors, with the collaboration of Bustarret, Lansade, and Limasset, and of Mme Huan for the coloured plates, give a full, semi-popular account intended to be of use to growers, of the chief potato diseases occurring in France (including *Erysiphe cichoracearum*, an undetermined bacterial wilt, and *Sclerotinia libertiana* [*S. sclerotiorum*]), their symptoms, manner of transmission, and control. A key to the different diseases is provided.

Limasset (P.) **Les maladies à virus de la Pomme de terre.** [Potato virus diseases.]— *Circ. Lig. nat. Lutte Ennem. Cult.*, 8 pp., May–June, 1941. [Received September, 1945.]

In this address, delivered on 15th May, 1941, in Paris, the author discusses in semi-popular terms the question of potato virus diseases, with special reference to their symptoms, effects, transmission, and control. The diseases dealt with include the mosaic group (virus Y, causing 'bigarrure' [streak] and 'chute des feuilles' [leaf-drop streak], virus A, virus X, and the virus causing 'mosaïque aucuba' or 'panachure infectieuse' [aucuba mosaic]), the 'enroulement' [leaf roll] group, which comprises only one virus, but the one that causes the most damage in France, the 'tubercule en fuseau' [spindle tuber] group, comprising two viruses confined to the American continent, and the 'jaunisses' [yellows] group, including the potato 'balai de sorcière' [witches' broom] virus, found in the United States and Russia.

Saha (J. C.). **Diseases of Rice and methods for their control.**—*Sci. & Cult.*, xi, 1, pp. 13–20; 2, pp. 69–74, 3 figs., 1945.

Of the rice diseases listed with descriptive notes in this summary of the main information available on the subject, the following are definitely known to occur in India: false smut (*Ustilaginoidea virens*), bunt (*Tilletia* [*Neovossia*] *horrida*), *Sclerotium oryzae*, *Helminthosporium oryzae* [*Ophiobolus miyabeanus*] (the virulent

epidemics of which in 1942 were a contributing factor in the Bengal famine of 1943), 'pan-sukh' [*R.A.M.*, xvi, p. 833], straighthead [ibid., xxi, p. 128], leaf smut (*Entyloma oryzae*), Udbatta disease (*Ephelis oryzae*), the agent of total destruction of the inflorescences, root rot (the 'mentek' of the Dutch East Indies [ibid., xvi, p. 339]), *Fusarium* sp., and blast (*Piricularia oryzae*).

The 'myitpo' disease, reported from irrigated tracts in the dry Shwebo district of Burma, appears in patches 30 to 100 ft. or more in diameter, on which the plants are stunted, yellow, and desiccated: its etiology is obscure, but a biological agency is suspected. Control may be effected by the application of soluble phosphatic fertilizers.

LINDEBERG (G.). **Über die Physiologie ligninabbauender Boden-Hymenomyzeten. Studien an schwedischen Marasmius-Arten.** [On the physiology of lignin-decomposing soil Hymenomycetes. Studies on Swedish species of *Marasmius*.]—*Symb. bot. upsaliens.*, viii, 2, 183 pp., 24 graphs, 1944. [English summary.]

The capacity of 14 species of *Marasmius*, mostly common occupants of the soil of Swedish hard- and softwood forests, for litter (chiefly lignin and cellulose) decomposition was investigated by culturing the fungi for six to seven months on sterilized pine needles, beech and aspen leaves, and *Glyceria maxima* straw, and determining the dry weight of the substrata and their contents of lignin, cellulose, and furfural-yielding substances at the beginning and end of the experimental period.

All the species except *M. chordalis* decomposed lignin more or less extensively, *M. putillus*, *M. scorodoneus*, *M. peronatus*, and *M. androsaceus* being the most active in this respect on pine needles (48 to 59 per cent.), *M. alliaceus*, *M. foetidus*, and *M. ramealis* on beech leaves (67 to 72 per cent.), and *M. oreades* on straw (63 per cent.). Cellulose was decomposed by all the species, some of which disintegrated 80 to 90 per cent. of the substance in all the substrata except pine needles; here the process was evidently more difficult, and in no case did the amount of disorganization exceed 36 per cent. All the species severely attacked the furfural-yielding components of the litter.

Nitrate and organic nitrogen were assimilable by all the species, but *M. fulvobulbillosus* was the only one capable of absorbing ammonium nitrogen.

CRANDALL (B. E.). **Nursery root and collar rot of Cinchona and root rot of Canavalia caused by Sclerotium rolfsii.**—*Plant Dis. Reptr*, xxix, 20, pp. 536–537, 1945. [Mimeographed.]

This paper records the isolation of a *Sclerotium* sp., possibly *S. rolfsii*, from dying Ledger type *Cinchona officinalis* transplants observed in the nursery. Scattered dying patches, associated with a cluster of dead or dying seedlings, were in obvious contrast to transplants killed by die-back or root rot (*Phytophthora* spp.). An examination of the soil surrounding the diseased trees disclosed abundant production of the sclerotia of the fungus. The elimination of diseased and adjacent trees and the removal of a portion of the bed surface are said to have checked the spread of the disease, which is believed to be the first record of southern blight (*S. rolfsii*) attacking *Cinchona* in any part of the world.

MARTÍNEZ (J. B.). **El Agárico blanco ('Ungulina officinalis' Pat.) en los Cedrales del Marruecos español.** [The white Agaric ('*Ungulina officinalis*' Pat.) in Spanish Moroccan Cedar plantations.]—*An. Jard. bot. Madr.*, iv, pp. 75–115, 12 pl., 1944. [German, French, English, and Italian summaries.]

Specimens of *Ungulina officinalis*, of which *Fomes officinalis* [*R.A.M.*, xxii. p. 231] is listed as one of the nine synonyms, were collected on *Cedrus atlantica*

in Spanish Morocco and subjected to a critical examination by the author, who differentiates four hyphal types within the fruit body, namely, ordinary hyphae (not staining with blue lactic acid), vermiform (reacting positively to the same stain), fibriform (giving a negative or faint response), and multiform (taking a vivid stain). The most abundant growth was made on potato dextrose agar at P_H 7, but satisfactory development also occurred on sour cherry juice agar at P_H 4. In view of the pharmaceutical importance of the fungus and its prevailing scarcity on the market, further investigations are planned to determine the possibilities of (*a*) its procurement from stands of *Abies pinsapo* in Spain, *A. maroccana* and *C. atlantica* in Spanish Morocco, and *Pinus canariensis* in the Canary Islands, and (*b*) artificial production by inoculation of the coniferous hosts.

RAMSBOTTOM (J.). **Reasons for name-change and the stability of names.**—*Ann. appl. Biol.*, xxxii, 2, pp. 181–183, 1945.

Opening a discussion on biological nomenclature [*R.A.M.*, xxiv, p. 166], the author remarked that stability in nomenclature is a desideratum, and that economic biologists appear to desire to prevent name-changes which, though sound in principle, are objectionable in practice. While the conservation of generic names is beneficial, there is no support by botanists for the proposal to conserve specific names. Absolute stability of nomenclature cannot be reconciled with taxonomic progress. Lists of standard names could not prevent changes based on new systematic knowledge, and would not be regarded as satisfactory by systematists.

BISBY (G. R.). **What are the mycologists doing about nomenclature?**—*Ann. appl. Biol.*, xxxii, 2, pp. 183–184, 1945.

After commenting on the undue length of the International Rules of Botanical Nomenclature, the author suggests that they could be somewhat simplified, and that it might be useful to have a shorter, supplementary code for fungi. The views of plant pathologists, industrial mycologists, and others, should be obtained regarding the capitalization of epithets and the possible conservation of important ones. The conservation of generic names should receive further consideration, and preparation begun for the proposal of important genera for conservation at the next Botanical Congress. Finally, the author recommends the reconstruction of the Special Committees appointed by Congresses, in order to secure more effective action.

JØRSTAD (I.). **Some Cyprian Uredinales.**—*Nyt Mag. Naturv., 1942*, 83, pp. 191–229, 1942. [Received September, 1945.]

Among the 25 rusts recorded from Cyprus in this paper [*R.A.M.*, xviii, p. 90] are *Gymnosporangium fuscum* (commonly referred to *Gymnosporangium sabinae* (Dicks.) Wint., an invalid binomial under the Rules of Botanical Nomenclature, the epithet being prior to 1801) found on *Pyrus syriaca*, and *Uromyces trifolii-repentis* on *Trifolium fragiferum* and *Hippomarathrum crassilobum* (the latter a new host for the rust). The name *U. trifolii* (Hedw. f.) Lév. is inadmissible, as *Puccinia trifolii* Hedw. f. ex DC. was recorded as occurring on *T. repens*, *T. filiformis* (= *T. minus* Sm.) and *T. hybridum*, and evidently embraces both *U. nerviphilus* (syn. *U. flectens*) and *U. trifolii-repentis* (but not the common rust on *T. pratense*, cf. *U. fallens*).

JØRSTAD (I.). **The Ustilagineous genus Tuburcinia in Norway.**—*Nyt Mag. Naturv., 1942*, 83, pp. 231–246, 1942. [Received September, 1945.]

Descriptive notes are given on 20 species of *Tuburcinia* found in Norway, with keys to the species found on each of the different host-families. In an introductory paragraph the author states that in his opinion the proposal by the Plant Pathological Committee and the Nomenclature Committee of the British Mycological

Society to place *Urocystis* on the list of Nomina Generica Conservanda [*R.A.M.*, xviii, p. 755] comes too late, as the renaming has already taken place, and the new names have already been used. Furthermore, many species described by Liro, Ciferri, Sydow, Lavrov, and Cunningham have no *Urocystis* epithet. To preserve *Urocystis* would, therefore, necessitate considerable renaming, and would also be of very doubtful advantage. The proposal should be rejected. If Liro, like Ulbrich, had held that it was justifiable to regard *Tuburcinia* and *Urocystis* as two distinct genera, the former embracing only one species, *T. orobanches* (Mérat) Fr. [ibid., xx, p. 598], no wholesale changing of names would have been necessary. *T. ferruginea*, described by Liro from tulip bulbs and later found in Norway on *Botrytis*-infested shallots and carrots, is now stated to belong to *Papulaspora*.

Gutner (L. S.). Головневые Грибы. [The smut fungi of the U.S.S.R.]—vi+374 pp., 137 figs., Leningrad, State Publishing Department, Section of Agriculture, 1941. [Received September, 1945.]

This work is based upon the uncompleted manuscript of the late A. A. Jaczewski, written during the years 1916 to 1920 and unpublished at the time of his death. It has been revised and brought up to date by the author, who has retained 140 of the 207 species embracing 10 genera described by Jaczewski, and has added 318 other species belonging to 23 genera, including 5 new species and 5 new combinations. The first part of the book (pp. 7–30) provides general information on the smuts and the latter part (pp. 31–331) deals with their systematic characters and position (including notes on geographical distribution). Some species have been included which, while they are not found in Russia, occur in countries contiguous with or near to the U.S.S.R. Appended to the work are a list of excluded species, a bibliography of over 550 titles, and indexes of hosts and parasites.

Fischer (G. W.) & Hirschhorn (Elisa). **The Ustilaginales or 'smuts' of Washington.**—*Bull. Wash. agric. Exp. Sta.* 459, 84 pp., 52 figs., 1945.

In the introduction to this bulletin the authors state that approximately one-fifth of the smuts reported for the entire North American continent are known to occur in the State of Washington and that 146 species of plants serve as hosts for these 51 species of smuts.

The smuts described are arranged (with keys) in four artificial groups: stem smuts, leaf smuts, inflorescence smuts, and gall-forming smuts. In addition to a description, the synonymy, hosts, and photographs of the infected host and spores are given for each species. Host and parasite indexes and a select bibliography conclude this useful publication.

Viégas (A. P.). **Alguns fungos do Brasil. II. Ascomicetos.** [Some fungi of Brazil. II. Ascomycetes.]—*Bragantia, S. Paulo*, iv, 1–6, pp. 5–392, 220 pl., 34 figs., 1944.

Included in this copiously annotated list of Brazilian Ascomycetes [cf. *R.A.M.*, xxiv, p. 207], besides a number of new genera and species, are *Capnodium musae* n.sp. on leaves of banana; *Taphrina pteridis* n.sp. on fronds of *Pteris*; *Sclerotinia ricini* on *Ricinus communis* described by Godfrey from Florida [ibid., vi, p. 526], *Taphrina deformans* on peach, *Dictyostomiopelta manihoticola* n.g., n.sp., and *Diaporthe manihoticola* Viégas (*Trab. Reun. argent. Agron.* 71, 1941) on cassava, and *Montagnella opuntiarum* Speg. on *Opuntia ficus-indica*. The contribution is fully illustrated and the bibliography comprises 200 titles.

Viégas (A. P.). **Alguns fungos do Brasil. III. Ustilaginales.** [Some fungi of Brazil. III. Ustilaginales.]—*Bragantia, S. Paulo*, iv, 12, pp. 739–751, 10 pl., 4 figs., 1944.

Included in this critically annotated list of Brazilian Ustilaginales are *Sorosporium reilianum* [*Sphacelotheca reiliana*] on maize, *S. sorghi* on sorghum and *Sorghum*

halepense, *Ustilago tritici* on wheat, *U. zeae* [*U. maydis*] on maize and *Euchlaena mexicana*, and *Polysaccopsis hieronymi* on *Solanum* sp. [*R.A.M.*, xxii, p. 454].

Ellison (B. R.). **Flagellar studies on zoospores of some members of the Mycetozoa, Plasmodiophorales, and Chytridiales.**—*Mycologia*, xxxvii, 4, pp. 444–459, 4 figs., 1945.

In this paper the author shows that a germinating spore of *Plasmodiophora brassicae* [*R.A.M.*, xxiv, p. 43] was observed with two actively beating flagella, confirming Ledingham's researches [ibid., xviii, p. 449] indicating that the zoospores of *P. brassicae* are biflagellate. With reference to the tendency of some authors to refer the members of the Plasmodiophorales to the Woroninaceae [ibid., xxiv, p. 74], the author points out the presence of two forms of flagella in some species, one a tinsel and the other a whip-lash type, and considers that this must be held to exclude *P. brassicae*, the type-genus and species of the family Plasmodiophoraceae, and the order Plasmodiophorales from relationship to the Woroninaceae on the ground of its type of flagellation.

Unamuno (L. M.). **Notes micológicas.** [Mycological notes.]—*An. Jard. bot. Madr.*, iv, pp. 145–166, 6 figs., 1944.

The following are among the 72 species of Spanish fungi (of which four are new) comprised in this critically annotated list [cf. *R.A.M.*, xx, p. 495, xxiv, p. 387]: *Septoria chrysanthemi-indici* on *Chrysanthemum maximum* [ibid., vii, p. 245]; *S. dianthi* on *Dianthus armeria*, a new record for the country; *Septogloeum carthusianum* on *Euonymus europaeus*; and *Alternaria hortensiae* n.sp., producing on the leaves of *Hydrangea hortensis* numerous circular lesions, 2 to 8 mm. in diameter, brownish-red on the lower and dark purple on the upper surface, with whitish centres, and characterized by brown, straight or slightly curved, bi- to triseptate conidiophores, united in fascicles of two or three, 40 to 72 by 7·5 to 8 μ, and brown, oblong to subclavate conidia, 48 to 70 by 15 to 17 μ, provided with 5 to 9 transverse septa and generally one longitudinal, oblique septum, and with a hyaline or subhyaline beak, 70 to 135 μ in length, having up to four septa.

Jordán de Urríes y Azara (M.). **Hongos microscópicos del Valle de Benasque (Pirineos aragoneses).** [Microscopic fungi of the Valley of Benasque (Aragonese Pyrenees).]—*An. Jard. bot. Madr.*, iv, pp. 167–193, 7 figs., 1944.

Included in this critically annotated list of 63 Pyrenean fungi are *Puccinia allii* on *Allium carinatum*, *P. buxi* on box (*Buxus sempervirens*), *Valsa ambiens* on *Corylus avellana*, and *V. leucostoma* on birch [*R.A.M.*, xxii, p. 42].

Losa España (D. M.). **Aportaciones a la flora micológica gallega.** [Contributions to the Galician mycological flora.]—*An. Jard. bot. Madr.*, iv, pp. 195–240, 23 figs., 1944.

Among the fungi (about 100) listed with critical annotations in this further contribution to the knowledge of Galician fungi [*R.A.M.*, xiii, p. 596], may be mentioned *Cylindrosporium cameliae* Gonz. Frag. on *Camellia* sp., *Cicinnobolus crepidis* n.sp. on *Crepis virens* leaves attacked by *Oidium erysiphoides*, *Puccinia antirrhini* on *Antirrhinum majus* (the first record for Spain [but see ibid., xxii, p. 53]), and *P. fusca* on *Anemone nemorosa*.

Rayss (T.). **Contribution à l'étude des Deuteromycètes de Palestine.** [Material for the study of the Deuteromycetes of Palestine.]—*Palest. J. Bot.*, J. Ser., iii, 1, pp. 22–51, 1 pl., 4 figs., 1943. [Received August, 1945.]

Continuing his studies of the mycoflora of Palestine [*R.A.M.*, xxii, p. 497], the author supplies a list of 150 species, of which the following may be mentioned:

Phyllosticta fusiformis f. *microcarpa* n.f. [ibid., vii, p. 652], on loquat leaves, characterized by pycnidia smaller than the typical species with walls only 25 μ thick and spores often containing droplets; *Cicinnobolus cesatii* [ibid., xxii, p. 264] and *C. taraxaci* on *Oidium* spp.; *Cytospora* [*Valsa*] *ambiens* [ibid., xxii, p. 42] strongly attacking poplar (*Populus nigra*); *Darluca filum* parasitizing *Puccinia antirrhini* so powerfully that it may be a means of controlling the rust; *Ascochyta pisi* on peas; *A.* [*Didymella*] *lycopersici* on frost-damaged potato leaves; *A. trifolii* on *Trifolium alexandrinum*; *Septoria divaricata*, with which *S. drummondi* [ibid., xx, p. 469] is considered by Grove [ibid., xv, p. 53] to be identical, on phlox; *S. iridis* found on leaves on *Iris haynei* but not on those of adjacent varieties; *S. lycopersici* on tomato; *S. petroselini* on parsley; *S. rubi* on leaves of *Rubus sanctus*, a new host; *Colletotrichum circinans* on shallots, a new record for Palestine; *Cercospora armoraciae* on horse-radish; *Cercospora judaica* n. sp. on vine leaves, producing scattered, round, amphigenous spots, 4 to 10 mm. in diameter, with dark margins, and characterized by densely fasciculate, straight or somewhat bent, dark olive, septate conidiophores, 27 to 45 by 4 to 6 μ, and straight or curved, pale olive, uni- to quadriseptate conidia, 32 to 50 by 6 to 8 μ; *C. personata* on groundnut; *Helminthosporium teres* on barley; *H. turcicum* on maize; *Tuberculina persicina* on various rusts [ibid., xxii, p. 264]; and *Isariopsis griseola* on bean leaves.

Zwirn (H. E.). **Studies on Leveillula taurica (Lév.) Arn.**—*Palest. J. Bot.*, J. Ser. iii, 1, pp. 52–53, 1943. [Received August, 1945.]

The author states that *Leveillula* [*Oidiopsis*] *taurica* is widely distributed in Palestine on tomatoes, eggplants, artichokes, and various wild plants [*R.A.M.*, xv, p. 683; xviii, p. 83]. It has also been found by Dr. Bremer on potato in Turkey.

In inoculations carried out with conidia from various hosts the fungus failed to pass from one genus to another, whereas the controls were readily infected provided the temperature was not too low or the atmospheric humidity too high. Biometrical measurements of conidia from different hosts showed marked variations, especially as regards the coefficient length/width, those borne on leaves floated on sugar solution being much larger than the normal ones, although the length/width ratio remained constant. The coefficient proved similarly stable in conidia from the same host species taken from the north and south of the country.

O. taurica is markedly xerophytic. Conidia germinate best at 26° C. on a dry slide with a relative humidity between 52 and 75 per cent. Infection does not take place through the stomata, as Blumer thought [ibid., ii, p. 181], but by means of an infection hypha derived from an appressorium. The influence of the parasite on the host consists mainly of a disturbance in the water relations, diseased leaves having a higher transpiration and a lower water content than healthy ones. Thus they shrivel and drop.

Clayton (E. C.) & Gaines (J. G.). **Temperature in relation to development and control of blue mold (Peronospora tabacina) of Tobacco.**—*J. agric. Res.*, lxxi, 4, pp. 170–182, 4 figs., 1945.

The inhibitory effects of moderately high temperatures on blue mould of tobacco (*Peronospora tabacina*) observed in Georgia led the authors [*R.A.M.*, xxiv, p. 251] to investigate the possibility of controlling the disease through temperature regulation. Attempts made to determine the optimum temperature for spore germination showed great variations, some collections germinating best at temperatures between 35° and 50° F. and others between 64° and 79°; a single optimum temperature for germination does not exist. As no germination took place at or above 85° or at 32° or below, the optimum temperatures embraced a range little less than the total range over which germination occurred. Leaf infection was

favoured by temperatures of 64° to 75° F. The spores are produced in the early morning and die in a few hours unless infection occurs, maximum sporulation occurring at a night temperature of 60° F. preceded by a higher day temperature. Viability of the spores was affected by the temperature at the time of formation and at germination. Minimum night temperatures of 70° F. and above almost inhibited sporulation in tobacco plant beds and their maintenance provided an effective control of mould disease, but plants grown in reduced light, as a result of shading or greenhouse-grown plants in midwinter conditions, sporulated freely at minimum temperatures as high as 80° F., and under such conditions temperature control is impracticable. The lethal effect of temperatures over 85° on the fungus was not modified by the conditions of the host, and blue mould in plants constantly exposed to reinfection was controlled by exposure to 110° for 4 hours per week. Complete control in greenhouse plants, using heat from the sun, was achieved at temperatures of 105° to 110°. Heat treatment, however, generally costs more than spray or gas treatment, and is not likely to be widely adopted.

Costa (A. S.). **Moléstias de virus do Fumo no Estado de S. Paulo.** [Tobacco virus diseases in the State of S. Paulo.]—*Bragantia, S. Paulo*, iv, 12, pp. 709–738, 26 figs., 1944.

A well-illustrated account is given of the symptoms and etiology of the following virus diseases of tobacco in São Paulo, with directions for their control: 'vira-cabeça' (tomato spotted wilt virus) [*R.A.M.*, xxi, p. 354], white necrosis or 'cabbage tobacco' (probably due to the streak virus of J. Johnson [ibid., xix, p. 497]), veinbanding [potato virus Y: ibid., xx, p. 606], tobacco and cucumber mosaics and (?) tobacco leaf curl [ibid., xix, p. 305].

Gerstel (D. U.). **Inheritance in Nicotiana tabacum. XX. The addition of Nicotiana glutinosa chromosomes to Tobacco.** —*J. Hered.*, xxxvi, 7, pp. 197–206, 3 figs., 1945.

This paper describes the addition of chromosomes from *Nicotiana glutinosa* to tobacco by the utilization of artificial polyploidy and a series of back crosses and selfings [*R.A.M.*, xxiii, p. 152]. One of the characters of *N. glutinosa* thus incorporated in tobacco was resistance to tobacco mosaic disease. The additional races proved meiotically rather stable and bred fairly truly.

Bryant (L. R.) & Kreutzer (W. A.). **Wet soil promotes Tomato rot.**—*Food Packer*, xxvi, 8, pp. 59–60, 1 fig., 1945.

In 1943 a trial of three cultural practices, viz. ridging, mulching, and staking, designed to prevent tomato rot (*Phytophthora capsici*) by the elimination of excessive moisture, an essential condition for its development, was set up at the Colorado Agricultural Experiment Station [*R.A.M.*, xxiv, p. 124]. These methods were compared with the ordinary custom of growing the plants on flat ground and allowing them to sprawl unhindered. The average percentages of healthy fruits in the ridged, mulched, staked, and control plots, which were heavily inoculated and irrigated at the onset of ripening, were, 80·78, 89·42, 98·05, and 60·53, respectively. In 1943 seven fungicides were tested on Landreth tomatoes grown under the most favourable conditions for the development of *P. capsici* with the following results (in average percentages of sound fruits): 5 per cent. yellow cuprocide plus 95 per cent. Cherokee clay, 82·10; same proportions of yellow cuprocide and talc, 83·6; 10 per cent. yellow cuprocide plus 90 per cent. talc, 77·82; 10 per cent. basicop plus 90 per cent. talc, 72·69; 10 per cent. tribasic copper sulphate plus 90 per cent. talc, 71·93; 10 per cent. copper oxychloride plus 90 per cent. talc, 82·37; Bordeaux mixture 4–4–50, 83·17; and control, 65·43. It will be seen from these data that

the yellow cuprocide and copper oxychloride dusts approximate in efficacy to Bordeaux mixture, and the former are much easier to apply.

In 1944 preliminary tests were carried out with a new procedure, involving the running of the irrigation water over 5 lb. copper sulphate for each 135 ft. of row immediately after inoculation of the soil. Although the prevailing temperatures were too low to permit of extensive spread of the pathogen, the results were sufficiently promising to justify further tests of the treatment.

Aberdeen (J. E. C.). **Diseases of the Tomato and their control.**—*Qd agric. J.*, lx, 5, pp. 277–299, 10 pl., 1945.

A key is given to aid in identifying tomato diseases in the seed-bed and field, followed by a description and recommendations for control of Irish blight (*Phytophthora infestans*), target spot (*Alternaria solani*), leaf spot (*Septoria lycopersici*), bacterial spot (*Bacterium vesicatorium*) [*Xanthomonas vesicatoria*: *R.A.M.*, xx, p. 152], *Fusarium* wilt (*F. bulbigenum* var. *lycopersici*), *Verticillium* wilt (*V. albo-atrum*), bacterial wilt (*Bacterium* [*Xanthomonas*] *solanacearum*), bacterial canker (*Aplanobacter* [*Corynebacterium*] *michiganense*), virus diseases, namely, mosaic, spotted wilt, big bud [ibid., xx, p. 182], streak, and fern leaf [ibid., xvii, p. 354], and blossom-end rot.

Costa (A. S.). **Observações sôbre vira-cabeça em Tomateiros.** [Observations on 'vira-cabeça' in Tomatoes.]—*Bragantia, S. Paulo*, iv, 8, pp. 489–507, 8 figs., 1944.

Much of the information presented in this study on tomato 'vira-cabeça' [a strain of the tomato spotted wilt virus] in São Paulo, Brazil [*R.A.M.*, xxiii, p. 365], has already been noticed from other sources, but the following points may be of interest. In one out of two varietal reaction experiments Market King contracted the lowest percentage of infection (3·6) and a type of Rei Humberto the highest (39·7), while in the other the minimum of 3·8 developed on Rutgers and the maximum of 14 on Seleto. Among other well-known commercial varieties included in both tests, Earliana, Lucullus, Bonny Best, Marglobe, and Stone showed 12·3, 13·8, 18·5, 25·5, and 26·4 per cent. spotted wilt, respectively, in the first, and 13·8, 12·6, 11·4, 7·8, and 12, respectively, in the second. *Lycopersicon pimpinellifolium*, *L. hirsutum*, and certain strains of *L. peruvianum* have shown resistance to spotted wilt in São Paulo, and the same is true of hybrids between the first-named and Early Jewel, the fruits of which, however, approximate to those of the wild parent and are thus without commercial interest.

'Vira-cabeça' is undoubtedly the most serious disease of tomatoes in some localities, e.g., at the Citricultural and Central Experiment Stations, Campinas, where entire crops may be involved, while a private grower in the same district was compelled in 1939 to abandon a planting too heavily infected to be worth the expense of picking.

Roguing for the control of 'vira-cabeça' should be carried out immediately the first symptoms appear; at a later stage it is of little use. In experiments on tobacco the extermination of the insect vector (*Frankliniella* sp.) in the seed-bed failed almost completely to reduce the incidence of the disease in the field, and presumably the same consideration would apply to tomatoes. An important measure of control may be achieved by the choice of suitable localities for tomato plantings, avoiding the humid conditions favouring an abundance of thrips. Another very effective means of combating 'vira-cabeça' in tobacco (and here again tomatoes would probably respond similarly) consists in transplanting at the beginning and end of the rainy periods, i.e., September to October and February to March, when the plants are likely to develop most satisfactorily.

Kleczkowski (A.). **Conversion of non-precipitating and inhibiting protein complexes into forms again precipitable by the antisera to the original proteins.**—*Brit. J. exp. Path.*, xxvi, pp. 33–41, 1945.

Experiments carried out by the author demonstrated that when the tomato bushy-stunt virus [cf. *R.A.M.*, xxiv, p. 292] was heated together with a serum albumin, the virus was able to combine with the albumin to form a complex which did not precipitate with the antiserum to the virus. The virus, which is resistant to peptic proteolysis, was recovered in the precipitable form by peptic hydrolysis of the complex.

Clauson-Kaas (N.), Plattner (P. A.), & Gäumann (E.). **Über ein welkeerzeugendes Stoffwechselprodukt von Fusarium lycopersici Sacc.** [On a wilt-producing metabolic product of *Fusarium lycopersici* Sacc.]—*Ber. schweiz. bot. Ges.*, liv, pp. 524–527, 1944.

Two distinct types of wilting developed in the detached pinnate leaves of six- to eight-week-old tomato plants, with petioles 8 to 10 cm. long, placed in filtrates of cultures of *Fusarium* [*bulbigenum* var.] *lycopersici* [*R.A.M.*, xiv, p. 310] on Richards's solution. (1) The petiole retained its erect habit and normal diameter, though becoming rather dry. The leaves showed black spots and assumed a brittle texture, curling upwards at the margins. These symptoms were characteristic of leaves immersed in a 0·1 per cent. solution of the filtrate adjusted to P_H 7 with a dilute sodium lye containing a trace of ferric chloride. (2) The petiole drooped, becoming soft and flaccid; the portion projecting from the solution eventually shrivelled into a fine thread. The leaves rolled sharply inwards, became very limp, and turned blackish-green to black; it was some time before they shrivelled and crumbled. This form of wilting was observed on leaves in the pure filtrates. Besides these two well-defined syndromes, various transitional types of the disease occurred on the inoculated material.

A leaf immersed in 7 c.c. water containing 2 mg. culture filtrate + 0·04 mg. ferric chloride (P_H 7) wilted in 36 to 60 hours, whereas in the absence of the iron activator the process took 100 to 150 hours. When leaves from five tomato plants that had been kept in a dark room for 15 hours were placed in a 0·03 per cent. filtrate solution with an admixture of ferric chloride, no wilting developed in 50 hours, whereas leaves taken from the identical plants after three hours in daylight and transferred to the filtrate, also in daylight, wilted completely during the same period. On removal to daylight, the leaves kept in the dark wilted in 40 hours. Loss of turgidity in the treated foliage would thus appear to be associated with active transpiration and a correspondingly heavy intake of water [cf. ibid., xi, p. 86].

Irving (G. W.), Fontaine (T. D.), & Doolittle (S. P.). **Lycopersicin, a fungistatic agent from the Tomato plant.** *Science*, N.S., cii, 2636, pp. 9–11, 1 fig., 1 graph, 1945.

From the expressed juice of Pan America tomato plants the authors obtained an antibiotic agent 'lycopersicin' which, though still impure, shows marked fungistatic activity towards *Fusarium bulbigenum* var. *lycopersici*. It occurs throughout the mature plant, and in the crude preparations available withstands autoclaving for at least 15 minutes at 15 lb. pressure. It is dialysable, adsorbed from aqueous solution at P_H 5·5 on charcoal, soluble in water and methanol, partially soluble in ethanol, and insoluble in chloroform, acetone, ethyl acetate, ether, petroleum ether, and benzene.

A rapid and accurate method for the assay of the fungistatic activity of lycopersicin, based on the cylinder-plate method, was devised. Growth of the fungus was uniformly raised, white, and cottony, developing evenly and spontaneously over the whole agar surface. Inhibition zones surrounding the cylinders were

sharply defined and susceptible of accurate measurement. The control cylinders, filled with water, showed no inhibition.

Preliminary assays of the different parts of a number of tomato varieties for lycopersicin activity at all stages of growth indicated that (*a*) all the varieties tested, including Bonny Best (highly susceptible to wilt), Rutgers and Marglobe (resistant), and Pan America and Red Currant (highly resistant) contain the inhibitor; (*b*) lycopersicin activity, absent from the seed, appears in seedlings germinated in the dark and in the plant within eight days after planting; (*c*) the concentration of lycopersicin varies to some extent with the age of the plant and considerably with the plant part assayed.

Ogilvie (L.). **Seed infection as a cause of outbreaks of Didymella stem-rot of Tomato.**—*Gdnrs' Chron.*, Ser. 3, cxviii, 3060, pp. 71–72, 2 figs., 1945.

A new and alarming feature of the tomato stem and fruit rot (*Didymella lycopersici*), prevalent in recent years in the west of England, is the occurrence of stem rot in glasshouses. After reviewing the researches of Orth, Sheard, and Small [*R.A.M.*, xviii, p. 636; xxii, pp. 378, 466], the author states that C. J. Hickman found that seven out of ten samples of seeds from the Evesham district were infected in 1942. In experiments at Long Ashton infected seed grown in sterilized soil gave rise to seedlings infected with *D. lycopersici*, 8·5 per cent. in one test and about 5 per cent. in another. The tips of the cotyledons were shrivelled and appeared to have been infected directly from the seeds, which were carried up above the soil. Later the badly diseased ones died off, while infected cotyledons on otherwise healthy plants dropped and the plants recovered.

In a further test seed disinfection with organo-mercury dusts eliminated infection in the seedlings, but later over 80 per cent. of the plants showed stem rot. It is concluded that infected seed can give rise to infected seedlings, some of which die off and infect the soil, leading to stem rot among older plants at soil-level. The gravity of this is only apparent when it is realized that stem-rot infection under such condition often becomes epiphytotic, particularly in glasshouses, without symptoms being present on seedlings. Attributing this mainly to the use of contaminated seed culled from field-grown diseased fruit, the author considers the employment of carefully selected seed from healthy plants grown under glass a sound measure of control both for indoor and outdoor crops.

Taylor (G. G.). **Bordeaux mixture, copper oxychloride, and copper oxide sprays for control of late-blight (Phytophthora infestans) of Tomatoes.**—*N. Z. J. Sci. Tech.*, A, xxvii, 1, pp. 9–13, 1 fig., 1945.

The serious (frequently total) losses from late blight (*Phytophthora infestans*) experienced by Auckland tomato-growers were reduced to less than 1 per cent. by regular applications (seven or eight) of 3–4–50 Bordeaux mixture at heavy volumes (up to 516 gals. per acre) in experiments from 1940 to 1944 on the Kondine Red and Adelaide Dwarf varieties. Copper oxychloride (cuprox) at 5 lb. in 100 gals. is comparable to Bordeaux in its fungicidal action, and being free from the deleterious effects on plant growth exerted by the latter, it results in larger yields. Copper oxide (perenox) occupied a position similar to cuprox in the one year in which it was used. Further work is necessary to determine the optimum spray dosages and spray volumes per acre applicable to commercial conditions.

Joffily (J.). **Ferrugem do Eucalipto.** [Eucalyptus rust.]—Bragantia, S. Paulo, iv, 8, pp. 475–487, 4 pl. (1 col.), 1944. [English summary.]

Nursery plants of *Eucalyptus citriodora* in the municipality of Itaguai, Rio de Janeiro, Brazil, were observed in January, 1943, to bear the very inconspicuous uredospores of a rust, which was identified a month later, when teleutospores had

developed on 20 specimens transferred to the laboratory of the Campinas Agronomic Institute, as a *Puccinia* and specifically assigned by G. B. Cummins and F. D. Kern to *P. psidii* [*R.A.M.*, xxiv, p. 353]. The *Uredo* stage of the rust was first reported on *Eucalyptus* from Brazil in a preliminary list of the fungi of Espírito Santo by Gonçalves da Silva in 1939 [ibid., xx, p. 36], and in conversation with B. Pickel the writer learnt that it had also been found on the same host in Pernambuco. *P. psidii* is widely distributed in several parts of the country on Myrtaceae, but this appears to be the first record of its occurrence on *Eucalyptus*. Infection by the rust involves the foliage, petioles, and new stem tissues and renders the seedlings unfit for transplanting. Of 13 species of *Puccinia* recorded on Myrtaceae, only one, *P. rompelli*, appears from a perusal of the relevant literature and the examination of herbarium material to differ appreciably from *P. psidii*.

TINT (H.). **Studies in the Fusarium damping-off of conifers. I. The comparative virulence of certain Fusaria. II. Relation of age of host, pH, and some nutritional factors to the pathogenicity of Fusarium.**—*Phytopathology*, xxxv, 6, pp. 421–457, 3 figs., 10 graphs, 1945.

At the University of Pennsylvania, Philadelphia, four standard fungi, namely, *Sclerotium bataticola* [*Macrophomina phaseoli*], *Rhizoctonia* [*Corticium*] *solani*, *Pythium ultimum*, and *Penicillium* sp., and monospore cultures of 25 *Fusarium* lines were tested for pathogenicity on seedlings of eight conifers, viz., *Pinus resinosa*, *P. sylvestris*, *P. nigra* var. *austriaca*, *P. ponderosa*, *P. banksiana*, *Abies concolor*, *Picea pungens*, and *Pseudotsuga taxifolia* from different parts of the United States [*R.A.M.*, x, p. 569; xx, p. 612; *et passim*]. In tests on *Pinus resinosa* seedlings in quartz sand approximately half of the *Fusarium* spp. reduced emergence significantly compared with the controls, and all but one caused heavy post-emergence losses, the significance of the exception being questionable. *M. phaseoli* did not lower emergence but caused high post-emergence losses; *C. solani* effected severe losses in both categories; and *Pythium ultimum* and *Penicillium* sp. did not significantly reduce emergence or cause post-emergence losses. Of the various methods tried inoculations in sterilized soil cultures yielded the most statistically accurate results. Using this procedure, a number of *Fusarium* lines equalled and occasionally exceeded in virulence *P. ultimum* and *C. solani*, causing reduced emergence and increased post-emergence losses. *M. phaseoli* caused the lowest emergence losses, but post-emergence attack was relatively high. *Pinus resinosa* and *Pseudotsuga taxifolia* were the most susceptible of the hosts both to pre- and post-emergence infection, while *Pinus ponderosa* sustained the least damage from the former phase of the disease and *Picea pungens* was the most resistant to the latter.

Although nursery losses should ordinarily be less extensive than those induced experimentally, conditions of comparable severity might arise in certain circumstances, in which event normally saprophytic or moderately parasitic species of *Fusarium* could well be responsible for reductions in emergence on the scale found in these tests.

In *Pinus resinosa* seedlings grown in quartz sand culture and inoculated with two lines of *F. arenaceum*, *F. vasinfectum*, *F. oxysporum*, *F. aquaeductum* var. *medium*, *F. sporotrichioides*, and the four above-mentioned standard fungi, the losses due to the more virulent species appeared relatively early and reached their maximum levels within some three weeks of emergence, whereas those induced by less pathogenic lines were delayed, though generally culminating within 30 days. In the case of *M. phaseoli*, the losses were low for the first six weeks, after which they rose to a level round 90 per cent. before 75 days. Resistance to invasion by *F. oxysporum* increased directly with age in 1- to 55-day-old seedlings of *P. resinosa*.

In autoclaved and unsterilized soil samples, emergence reductions of *P. resinosa*,

P. ponderosa, and *P. sylvestris* due to inoculation with *F. oxysporum* and *F. avenaceum* were usually low to moderate at P_H values in the acid range [cf. ibid., xviii, p. 358] and high above P_H 8. Post-emergence damping-off was low in the more acid samples, reached a maximum on the acid side of neutrality, declined sharply in the sterilized soil at P_H 7·5 and in the unsterilized at P_H 8·1, and attained a uniformly high level in the most alkaline substrata. In quartz sand cultures of varying P_H, losses among *P. nigra* var. *austriaca*, *P. resinosa*, and *P. sylvestris* seedlings inoculated with the same two fungi were generally high at all acidity levels, though damping-off tended to become more prevalent with increasing alkalinity.

In quartz sand culture tests on the effects of variations in the concentration of certain components of the nutrient solution [ibid., ii, p. 82], a deficiency of nitrogen produced relatively negligible losses in *P. resinosa*, whereas excess caused early and severe infection by *F. oxysporum* and *F. avenaceum*. Calcium and phosphorus deficiencies resulted in heavy losses from damping-off, which were considerably reduced by an excess of these elements. Deficiencies of potassium, sulphur, and magnesium produced rather larger stands than the corresponding excesses, the latter series resembling controls grown and inoculated in standard solutions.

KIMMEY (J. W.). **The seasonal development and the defoliating effect of Cronartium ribicola on naturally infected Ribes roezli and R. nevadense.**—*Phytopathology*, xxxv, 6, pp. 406–416, 2 graphs, 1945.

The observations herein described and tabulated on the development and defoliating action of white pine blister rust (*Cronartium ribicola*) on *Ribes roezli* and *R. nevadense*, the principal associates of sugar pine [*Pinus lambertiana*], in their native habitat in the northern Sierra Nevada, California, were made in 1938, 1941, and 1943. In general, the susceptibility of the two species to infection and the capacity of *R. nevadense* for teleutospore production agreed with reports of earlier studies in British Columbia and the Pacific Northwest [*R.A.M.*, xii, p. 407; xvii, p. 639; xxiii, p. 122]. *R. roezli* remained the more susceptible, its infected leaf area producing a higher percentage of teleutosorus-bearing surface and nearly twice the amount of teleutosorus columns per unit of such surface, but the premature shedding of its diseased leaves still rendered it less of a menace to pines than *R. nevadense* per unit of population. In unworked areas of the sugar pine region, on the other hand, the larger population of *R. roezli* (2,900 ft. living branchwood per acre compared with 420 for *R. nevadense*) more than counterbalances its limited capacity for the retention of teleutosorus-bearing leaves, and here it is likely to prove a richer source of inoculum than *R. nevadense*.

Factors contributing to the premature shedding of infected leaves of *R. roezli* include severity of infection, relatively dry sites, youth and vigour of the foliage, exposure to direct sunlight, and early autumn frosts, of which only the first and last appreciably affected the process in *R. nevadense*. Infected *R. roezli* plants growing under shade and forming a profusion of new woody growth, especially on moist sites, are the most potentially dangerous in respect of the spread of blister rust, since the apical leaves of the current season's growth on such plants were the last to drop in the autumn and consequently bore uredospores and viable teleutosori for a maximum period.

The success or failure of *C. ribicola* to infect pines in any given year depends largely on the duration of rainfall before *Ribes* defoliation. Under infection and weather conditions comparable to those in the areas under observation in any of the three test years, *R. roezli* should bear sufficient inoculum for an attack on pine from late July to late September (or mid-October in the case of the shade forms), the corresponding period for *R. nevadense* extending from early August to late October.

Martínez (J. B.). **Un 'Fusarium' patógeno, nuevo paro la micoflora española.** [A pathogenic *Fusarium* new to the Spanish mycoflora.]—*An. Jard. bot. Madr.*, iv, pp. 61–73, 2 pl., 1944.

Fusarium oxysporum var. *aurantiacum*, a new record for Spain, is reported as the agent of damping-off of *Pinus pinea* in Alicante and *P. halepensis* in Valencia, both new hosts for science. Directions are given for the control of the disease by means of soil and seed disinfection, singly or combined, on the lines practised in the United States, Germany, and the U.S.S.R.

Rennerfelt (E.). **Der Zuwachs einiger Torulopsidacéen in Nährböden und Flüssigkeiten verschiedenen osmotischen Wertes.** [The growth of certain Torulopsidaceae in media and liquids of varying osmotic value.]—*Acta Hort. Gotoburg.*, *1940–1*, xiv, pp. 87–97, 2 figs., 3 graphs, 1940. [Received August, 1945.]

Certain members of the Torulopsidaceae isolated from mechanical wood pulp at the Gothenburg (Sweden) Botanical Institute [*R.A.M.*, xxi, p. 58], viz., *Geotrichum candidum*, *Mycotoruloides* sp., *Rhodotorula glutinis*, *R. gracilis*, *Torulopsis candida*, and *T. molischiana*, were grown on synthetic solid and liquid media adjusted to varying osmotic pressures by the addition of cane sugar. None of the organisms was able to develop on agar at a sugar concentration exceeding 60 per cent. (93·6 per cent. relative vapour tension), but in liquid media scanty growth was made at 80 (88 per cent.). The stronger the osmotic pressure of the substratum or the lower its relative vapour tension, the longer is the period required by the fungi for germination and the smaller are the colonies produced. These occupants of wood pulp were found to belong to the hygrophile group of fungi.

Gram (E.). **Klornitrobenzol-Forbindelser som Middel mod Kaalbrok, Kartoffelskurv, Kløver-Baegersvamp og 'Brune Rødder' paa Tomat.** [Chlornitrobenzene compounds as remedies against Cabbage club root, Potato scab, Clover rot, and 'brown roots' of Tomato.]—*Tidsskr. Planteavl*, xlix, pp. 119–143, 5 figs., 1 graph, 1944. [English summary.]

A tabulated survey is given of experiments in plant disease control in Denmark with a number of chlornitrobenzene preparations of I.G.Farben. Promising results were obtained in the control of turnip club root (*Plasmodiophora brassicae*) in 1935 by soil treatment with a mixture of brassicol (20 per cent. pentachlor-nitrobenzene +80 per cent. filler) [*R.A.M.*, xx, p. 33] and calcium nitrate at dosages of 500 and 525 kg. per ha., respectively, which increased the yield (in hectokg. per ha.) of sound or slightly infected roots from 10·8 for calcium nitrate alone and 21 for calcium cyanamide to 102·9. In 1941 the weight per white cabbage plant in plots treated with brassisan [loc. cit.; ibid., xxi, p. 177] at 30, 40, and 60 gm. per sq. m. amounted to 463, 530, and 730 gm., respectively, the corresponding figures for Bayer 2326 (a chlornitrobenzene compound of undisclosed composition) at the same rates being 700, 825, and 650 gm., respectively, and for the controls 317. The lowest incidence of club root (2·8 in a scale ranging from 0 to 5) occurred in the Bayer 2326 (30 gm.) plots.

In the case of potato scab (*Actinomyces scabies*), Bayer P (100 per cent. pentachlornitrobenzene) was the most effective of the fungicides tested, the percentages of almost or entirely scab-free tubers in the plots receiving 0, 60, and 120 kg. P per ha. in 1940 being 25, 61, and 69, respectively, and its action persisted the following year. In another trial in 1940 on a site already treated in 1939, the tuber and dry matter yields (in hectokg. per ha.) in the control plots and in those given 60 and 120 kg. P per ha. were 358, 379, and 384 and 84·6, 89·5, and 91·8, respectively. The scab index figures (0 to 3 scale) were 1·7, 1·3, and 1·1, the percentages

of almost or entirely scab-free tubers 48, 59, and 69, respectively, and the percentages of those bearing *Rhizoctonia* [*Corticium*] *solani* sclerotia 34, 17, and 15, respectively. In 1941 the scab index figure in the P plot receiving 60 kg. P in 1940 (nothing in 1939) was 1·4 as against 2·3 in the control and the percentage of almost or entirely scab-free tubers 57 compared with 20. The corresponding figures for the plot given 60 kg. P in 1939 (nothing in 1940) were 1·9 and 39, respectively. In a test in 1939 the joint application of P at 60 or 120 kg. per ha. and ammonium sulphate (500 kg.) gave very satisfactory results, the percentages of completely sound tubers in the plots receiving the two dosages being 45 and 54, respectively, compared with 27 in the control, and the scab index figures 0·9 and 0·8, respectively, as against 1·5.

Dusting with P and brassicol, with or without talc (1 : 4), effectively combated clover rot (*Sclerotinia trifoliorum*). For instance, in a test in 1939–40, the number of dead plants in plot of 3 by 3 m. dusted four times with brassicol-talc (100 kg. per ha.) was 2·2 compared with 13·2 in the control plot and 1 in that treated with brassicol (20 kg.). Using a portable rotation duster, one man can cover 1 ha. seed-clover in three hours.

Brown root rot is a glasshouse tomato disorder of obscure etiology [ibid., xviii, p. 715], characterized in the early stages by a zonate, brown discoloration of the fibrous roots which finally involves the entire system and paves the way for decay by facultative fungal and bacterial parasites. The premature destruction of the roots cuts off the water supply to the aerial organs, resulting in poor development, wilting, and a reduction of yield to half or a third of the normal. Some degree of control was achieved by the application of P or brassicol to the soil just before planting.

Samuel (G.) & Garrett (S. D.). **The infected root-hair count for estimating the activity of Plasmodiophora brassicae (Woron.) in the soil.**—*Ann. appl. Biol.*, xxxii, 2, pp. 96–101, 4 pl., 1945.

The authors present in this paper a method for estimating the relative numbers of *Plasmodiophora brassicae* spores germinating in different soils by counting infected root hairs, in the hope that it may yield results unobtainable by the growing of plants in pots or in the field, and valuable for the investigation of particular problems concerning club-root disease. Cabbage seedlings are grown for a week at 25° C. in glass tumblers of the infected soils; after washing out, the tap-roots are stained in 1 per cent. aceto-carmine and a count is made of the number of root hairs containing zoosporangia of *P. brassicae* along 2 cm. of root. The main factor inhibiting infection was found to be soil alkalinity, however produced. It was observed that in soils receiving N/10 sodium and potassium chlorides in place of distilled water the number of infected roots was reduced. Root-hair infection was also inhibited by low soil moisture content.

Walker (J. C.) & Hooker (W. J.). **Plant nutrition in relation to disease development.**—*Amer. J. Bot.*, xxxii, 6, pp. 314–320, 2 figs., 1945.

Young cabbage plants of (1) a strain highly susceptible to yellows (*Fusarium oxysporum* f. *conglutinans*) [*R.A.M.*, xxiii, p. 371], (2) an intermediate resistant strain, and (3) a highly resistant strain, growing in quartz sand cultures irrigated with nutrient solutions varying in salt concentration and in balance of nitrogen, phosphorus, and potassium were studied with a view to establishing the rate and severity of development of the disease. Except at low concentrations of inoculum, increase of salt concentration promoted a progressive decline in the rate of disease development, although slower at 19° C. than at 25°. While the disease development curves had the same relation to each other at 25° as at 19°, they were closer together after 20 days at the higher temperature. In the case of intermediate

resistant plants, inherent host resistance completely controlled the disease at 19° and no effect of salt concentration could be measured; while at 25° the curves for four out of five nutrient concentrations followed closely those for the susceptible strain at 19°.

Omission of potassium from the solution increased the rate of disease in the susceptible strain at 19° and 25°, but when nitrogen and phosphorus were omitted disease development decreased at 19° and 25° in the susceptible strain and at 25° in the intermediate resistant. The highly resistant strain showed no sign of disease whatever concentrations of salt or balance of nitrogen, phosphorus, or potassium were used in the nutrient. No correlation could be determined between the rate of growth of the host and the decline or increase of yellows infection brought about by changes in salt concentration or mineral balance.

GRAM (E.). **Virus-gulsot hos Beder. Beta Virus 4.** [Virus yellows of Beets. *Beta* virus 4.]—*Tidsskr. Planteavl*, xlvii, pp. 338–362, 1942. [English summary.]

Virus yellows of beets [sugar beet yellows: *R.A.M.*, xix, p. 386] has probably been in existence in Denmark at least since 1914. The disease chiefly affects the fodder varieties, those grown for sugar sustaining less damage. Sweet [? Swiss] chard (*Beta cicla viridis*) [? *B. vulgaris* var. *cicla*], red garden beets, and spinach are occasionally attacked. During the years 1932 to 1934, inclusive, infection occurred early and covered wide areas, and in such cases losses may be considerable, involving a fall of up to 3 per cent. in the sugar content and a 25 per cent. reduction in yield: in most seasons, however, the development of the virus is only sporadic.

Foliar yellowing is not a very reliable diagnostic feature, since it may arise from several causes and be more or less suppressed by shade, high precipitation, and the liberal use of nitrogenous manures. A brittle texture of the leaves is a valuable field symptom where the alternative possibility of magnesium deficiency can be excluded. Sachs's iodine starch test usually evokes a positive reaction before the appearance of the yellow discoloration, and shortly prior to, or coincidentally with, the development of brittleness in the foliage. Sugar beets sown in late autumn for seed production in the following year commonly escape infection, but fresh outbreaks of yellows may be caused by the visits of aphids to sprouting beets in open pits in the spring. The incubation period of the virus is about three weeks. Control measures [cf. ibid., xxiv, p. 397] should include the timely sorting of healthy mother-beets (before the onset of the normal autumn colouring), late sowing and early clearing of autumn spinach, and the location of first-year crops at a distance from seed beets or spinach exceeding the 500 m. prescribed as reasonably safe in the case of mosaic.

MILTHORPE (F. L.). **The compatibility of protectant seed dusts with root nodule bacteria.**—*J. Aust. Inst. agric. Sci.*, xi, 2, pp. 89–92, 1 fig., 1945.

Tests in which pea seed was simultaneously inoculated with *Rhizobium leguminosarum* [*R.A.M.*, xxi, p. 511] and treated with ceresan, cuprox, spergon, and chloranil showed that spergon alone allowed satisfactory nodulation, and even in this case the seed had to be sown within two hours of inoculation. Chloranil came next in order of successful nodulation, followed by cuprox, and then by ceresan. The last-named prevented all nodulation unless the seed was sown immediately after inoculation. It would seem that the ability of the nodule bacteria to diffuse away from the zone of toxic concentration of a protectant dust exercises an important effect on nodulation. The moisture content of the soil at sowing time would appear therefore to influence the degree of nodulation.

Wilt-resistant canning Pea named for Prof. E. J. Delwiche.—*Canner*, ci, 15, p. 11, 1945.

The first and only variety of canning pea bred for resistance to both common pea wilt [*Fusarium orthoceras* var. *pisi*: *R.A.M.*, xxii, p. 87] and near-wilt [*F. oxysporum* f. 8; ibid., xviii, p. 777] has been produced at the University of Wisconsin and is named Delwiche Commando. A sweet, mid-season variety with wrinkled seed, it has proved almost completely resistant to both diseases locally. Near-wilt has already been identified in specimens from Maryland, Delaware, New Hampshire, Massachusetts, Idaho, and Montana.

Cass Smith (W. P.). **The Bean mosaic menace.**—*J. Dep. Agric. W. Aust.*, xxii, 1, pp. 20–25, 3 figs., 1945.

A vegetable disease survey undertaken in April and May, 1944, disclosed widespread damage to bean crops attributed to the bean mosaic virus, involving a failure of 20 to 50 per cent. in an area including Wanneroo, Balcatta, Osborne Park, and Spearwood. The insidious progress of the disease is such that most growers were unaware of it as the agent of their losses and, in view of its being seed-borne and liable to dissemination by aphids, steps are being taken to produce disease-free seed from the healthiest crops of good type under Government supervision, and to familiarize growers with the symptoms of the disease and the measures required for its control.

The recent rapid spread of bean mosaic virus is explained by the scarcity of sound seed during the war. In these circumstances, growers have had to raise their own seed with a consequent fall in quality, the ill effect of which has been increased by failure to rogue out diseased plants and, in view of the high prices, by reserving for seed production the inferior 'tail-end' part of the crop—a practice which cannot be too strongly condemned, for the amount of infected seed may be seriously increased when only the late-maturing portion of the crop is saved. The recent considerable rise in the price of pole bean seed should simplify the production of disease-free seed under Government supervision.

Meanwhile, growers are recommended to raise their own stocks only from the best available seed, on plots isolated to prevent the spread of infection caused by aphid invasions from neighbouring affected bean crops; and as aphids thrive best in the cooler spring months, the planting of the seed crop should be delayed until early summer. Roguing-out should begin as soon as the first trifoliate leaves appear and be repeated once a week for the first month, and thereafter at fortnightly intervals throughout the growing season. To facilitate roguing of 'white-seeded runner' or other pole bean varieties the plants should be trained up single poles set vertically in the ground and spaced a little wider in the rows than usual. Any plants affected with halo blight [*Pseudomonas medicaginis* var. *phaseolicola*] or other diseases should be removed when roguing. Finally, on the crop reaching maturity, seed should be selected from good-type, heavy-yielding plants and, in the case of the 'white-seeded runner' variety, off-type plants bearing flat instead of round pods should be discarded.

Weber (Anna). **Mosaiksyge i Skalotter.** [Mosaic disease among Shallots.]—Reprinted from *Gartnertidende*, *1943*, 29, 2 pp., 1 fig., 1943. [Received August, 1945.]

Mosaic (*Allium* virus 1) [yellow dwarf] is reported to be so prevalent among shallots [*R.A.M.*, xxiv, p. 133] in Denmark that in some districts inspectors have been unable to find a single market-garden without some diseased plants. Steps have been taken in Jutland to procure healthy sets from gardens with a high proportion of sound plants, but the disease can only be controlled by the adoption

of similar measures on a wider scale. Under present conditions the crop is assuming greater importance than in former years, when yellow dwarf was largely overlooked, firstly because its infectious nature was not recognized, and secondly on account of the abundant supplies of healthy and relatively cheap onions available from southern countries.

White (N. H.). **Fungal soft rot of Carrots.**—*Tasm. J. Agric.*, xvi, 2, pp. 59–60, 1945.

Towards the end of May, 1944, carrots growing in the Smithton swamps of Tasmania rotted from the tips and the lowermost parts of the roots. The outside of the affected areas was depressed and frequently blackened, and the soft, water-soaked tissue underneath showed the presence of *Phytophthora megasperma* [*R.A.M.*, xiv, p. 211]. Inoculations of healthy, unwounded and wounded carrots with cultures from diseased material gave negative results. Dowson had obtained a similar result in 1931 [loc. cit.] and concluded that abnormally wet conditions were primarily responsible for predisposing the carrots to attack by the fungus. Rainfall records showed that both 1931 and 1944 were years of heavy May rainfall.

Fresh, healthy, mature carrots were then immersed in tap water for three days, when symptoms of 'drowning' set in, i.e., the cortical tissue became flaccid and there were indications of alcoholic breakdown. One lot of 'drowned' carrots was inoculated by placing the fungus on the surface or in a wound, and the remainder were left uninoculated. carrots that had not been 'drowned' being similarly treated, and all the lots kept in a moist atmosphere. After ten days at room temperature the 'drowned' inoculated carrots alone developed infection.

It is concluded that 'drowning' resulting from waterlogging in late autumn predisposed the plants to attack. Carrots grown in swampy soils should be dug and marketed as soon as mature, in early April, and adequate drainage should be provided.

Rader (W. E.). **Ansatospora acerina found causing decay of stored Carrots in Wayne County, New York.** –*Plant Dis. Reptr*, xxix, 20, p. 522, 1945. [Mimeographed.]

Ansatospora acerina [*R.A.M.*, xxiv, p. 305] was isolated from carrots collected during the winter and spring of 1945 from 20 storage plants in five counties of New York State in the course of an investigation conducted to ascertain the cause of an outbreak of decay in carrots which occurred in six storage houses in Wayne County, where decay of celery induced by *A. acerina* is highly prevalent. The fungus causes black lesions, indistinguishable from those produced by *Alternaria radicina*, on either the crown or the sides of the carrots. In cross section the rot produced by *Ansatospora acerina* is black except for the advancing margin, which is brown and usually presents a saturated appearance, in contrast to the discrete, black margins of the lesions formed by *Alternaria radicina*.

Tyner (L. E.). **Fusarium sambucinum Fkl. F. 6 Wr. as a pathogen of some species of the Cucurbitaceae.**—*Sci. Agric.*, xxv, 9, pp. 537–541, 1945.

During August, 1942, *Fusarium sambucinum* f. 6 was isolated from the stems of vegetable marrow plants at Brooks, Alberta, which had developed a severe wilt, accompanied by an extensive buff-coloured dry rot of the crown.

When cucumber seeds, both untreated and treated with 1 per cent. by weight of 2 per cent. ceresan dust were planted in pots in the greenhouse in five different samples of local soil inoculated with the fungus, the average percentage emergence of the treated and untreated seed was, respectively, 16·4 and 5·4, 8 and 8·4, 17·4 and 20·4, 38 and 6·4, and 18 and 18, as against 94 per cent. germination for the same seed in a moist chamber.

Cucumber, pumpkin, squash, and marrow seed both untreated and ceresan-dusted were planted in pots of inoculated soil in the greenhouse. Fifteen days later, the treated seed showed 40, 9, 70, and 10 per cent. living plants respectively, and the untreated 17·5, 0, 30, and 0. When cucumber, muskmelon, marrow, and pumpkin seeds were planted in pots of soil inoculated with cultures of the fungus from lucerne, potato, and marrow, only the marrow strain showed pathogenicity, which was very marked. The fungus showed increasing virulence to cucumber and pumpkin at temperatures ranging from 15° to 25° C.

In another experiment pumpkin, squash, and marrow seeds were planted on 19th May in 12 ft. rows in the field (25 seeds per row) and 150 gm. soil-grown inoculum added; by 5th July there were 76, 34, and 43 living plants, respectively, in the control rows and only 2, 8, and 0, respectively, in the inoculated. When the inoculum was placed in contact with the seed, only a few plants escaped destruction even in the seedling stage. Growth of the mycelium through the soil was demonstrated. This appears to be the first record of a physiologic form of this fungus being a major pathogen to cucurbits.

Walker (M. N.). **Galls on the roots of Citron-Watermelon hybrids.** -*Phytopathology*, xxxv, 6, pp. 480–482, 1 fig., 1945.

In 1933 and 1935 the roots of many plants of F_1 hybrids from watermelon-citron crosses obtained from the Iowa Agricultural Experiment Station and grown at Leesburg, Florida, were found on pulling to bear pedicellate, glistening white, later darkening galls, which on expansion tended to become tuberculate and divided, finally developing into dense, cauliflower-like structures entirely different from those associated with nematodes (*Heterodera marioni*) or *Bacterium tumefaciens*. Root proliferation round the galls was another feature of some of the excrescences. An inoculation experiment with the bacteria filling the intercellular pockets throughout the superficial layers of the galls gave inconclusive results, and no opportunity has yet arisen for further studies on the disorder.

Yu (T. F.), Chiu (W. F.), Cheng (N. T.), & Wu (T. T.). **Studies on Pythium aphanidermatum (Edson) Fitz in China.**—*Lingnan Sci. J.*, xxi, 1–4, pp. 45–62, 2 pl., 1 graph, 1945.

Since 1932 a destructive fruit rot of the white gourd (*Benincasa hispida*), prevalent in Kiangsu Province, has been under investigation at the Nanking College of Agriculture and at Chengtu in western China. The causal organism, *Pythium aphanidermatum*, besides attacking fruits and vegetables in the field, during storage, or in the market, is also responsible for damping-off of seedlings and decay of the vegetative organs. In the course of the studies herein described, the fungus was isolated from the following plants in addition to *B. hispida* [cf. *R.A.M.*, xiv, p. 7]: watermelon, melon, cucumber, vegetable marrow, squash, *Lagenaria leucantha*, *Luffa acutangula*, *L. cylindrica*, *Momordica charantia*, cotton (*Gossypium hirsutum* and *G. indicum*), chilli, tobacco, eggplant, sweet potato, bean, beet, Chinese cabbage, *Pachyrrhizus tuberosus*, *Ipomoea reptans*, *Amaranthus tricolor*, and *Aleurites fordii*, of which the four last-named are new hosts. The results of cross-inoculation experiments did not reveal any consistent differences in pathogenicity among the various isolates.

The development of *P. aphanidermatum* is promoted by a high relative humidity (90 per cent. and upwards) and retarded at or below 85 per cent. The optimum air temperature for the fungus lies between 30° and 35° C. Satisfactory growth was made on most standard media, the minimum, optimum, and maximum temperatures for mycelial expansion being 5° to 10°, 29° to 31°, and upwards of 41°, and the optimum for sporangial germination 24° to 26°. The optimum P_H

reaction for the growth of the pathogen is about 6·1, development ceasing at 2·5 and 10·7 on the acid and alkaline sides of neutrality, respectively.

P. aphanidermatum was isolated from various types of soil, including flower, vegetable, and fruit beds and forest ground (small conifers); in heavily infested field soil it was found at a depth of at least 8 in. below the surface in abundance varying in proportion to the amount of moisture present. Infection of field plants takes place exclusively through the soil, in which the fungus can persist for at least four years in a viable condition. Thus, cucumber seeds sown in soils one, two, three, and four years after infestation gave rise to seedlings with an average (in four replications) of 70·8, 17·9, 3·5, and 0·7 per cent. damping-off, respectively. Control measures, apart from the avoidance of continuous cropping, have not yet been worked out.

Wilson (C.). **A Diplodia associated with concealed damage in Peanuts.**—*Phytopathology*, xxxv, 6, p. 480, 1945.

'Concealed damage' is the term applied to an internal deterioration of Alabama groundnut seed characterized in the early stages by a small, dirty white to pale yellow spot on the inner face of the cotyledon which spreads until the whole embryo is blackened and pervaded by hyphae, while the cotyledons become rancid and are frequently intersected by a dense mycelial mat. Only the final stages of disorganization are apparent in unbroken seeds. The results of an examination in 1944 of over 1,200 seeds from samples collected in twelve widely separated fields in the south-east of the State do not support B. B. Higgins's conclusion that soil-inhabiting species of *Rhizoctonia*, *Penicillium*, and *Rhizopus* and *Sclerotium bataticola* [*Macrophomina phaseoli*] are responsible for the condition. The predominant organism obtained, irrespective of the method of sterilization or the medium used, was a *Diplodia*, tentatively identified as *D.* [*Botryodiplodia*] *theobromae*, which developed from 65 per cent. of all the seeds plated and constituted roughly 95 per cent. of the total number of isolates, *M. phaseoli*, *Fusarium*, *Aspergillus*, *Penicillium*, and *Rhizopus* spp. and bacteria accounting for the rest. The frequency of isolation of the *Diplodia* strongly suggests its etiological implication in the disease.

Viégas (A. P.). **Alguns fungos da Mandioca. I. II.** [Some Cassava fungi. I. II.]—*Bragantia, S. Paulo*, iii, 1, pp. 1-19; 2, pp. 21-29, 6 pl., 5 figs., 1943. [Received September, 1945.]

The cassava mildew occurring in Minas Gerais, Brazil, was referred by Müller to *Oidium manihotis* Áverna-Saccá [*R.A.M.*, xiv, p. 87], while Gonçalves da Silva assigns the species on the same host in Espírito Santo [ibid., xx, p. 36] to *O. manihotis* P. Henn. The author could not obtain the original diagnosis of either species, nor did he have access to Hennings's type material for comparison with the mildew on cassava in São Paulo, which can therefore only be provisionally identified with *O. manihotis* P. Henn. The conidia of the author's collections are larger (28 to 40 by 12 to 20 μ) than given by Saccardo in *Syll. Fung.* for *D. manihotis* P. Henn. The disease is without economic importance.

The collar rot caused by *Sclerotium rolfsii* may be responsible for heavy losses under the optimum conditions for the pathogen prevailing from December to February. Other hosts of the fungus in São Paulo include potato, *Boehmeria nivea*, bean, groundnut, *Crotalaria juncea*, Texas cotton, lucerne, carrot, strawberry, maize, rye, and the weeds *Oxalis* sp. and *Solidago microglossa*.

Phyllosticta manihotae n. sp., the agent of amphigenous, zonate, indistinctly marginate, often confluent lesions, ashen-grey on the under and brown to fuscous on the upper side, on living cassava leaves, is characterized by globose, subcuticular, fuscous pycnidia, aggregated or solitary, concentrically arranged, 100 to 170 μ

in diameter, shining ostioles, 15 to 20 μ in diameter, hyaline conidiophores, 1·5 to 2 μ in diameter, and hyaline, smooth, elliptic-ovoid conidia, 5 to 8 by 2 to 3 μ.

VIÉGAS (A. P.). **Pleophragmia manihoticola n. sp.**—*Bragantia*, iii, 3, pp. 45–48, 1 fig., 1943. [Received September, 1945.]

Pleophragmia manihoticola n. sp., isolated from lesions on cassava leaves at Campinas, São Paulo, Brazil, differing somewhat from those caused by *Cercospora henningsii* in the same State [*R.A.M.*, xx, p. 445], is characterized by sparse, obclavulate, short-stipitate, thick-walled, blunt-tipped asci, 100 to 180 by 40 μ, containing eight irregularly disposed, oblong-elliptical, fuscous, smooth, muriform ascospores, with a hyaline outer wall, divided by five transverse septa and one longitudinal septum, 20 to 24 by 7 to 9 μ. Paraphyses were not observed.

ARNAUD (G.). **Développement des maladies de la Vigne dans la région parisienne (mildion, Oidium, anthracnose).** [The development of Vine diseases in the region of Paris (mildew, *Oidium*, anthracnose)].—*Ann. Epiphyt.*, N.S., vi, 1, pp. 37–66, 7 graphs, 1940. [Received September, 1945.]

Observations carried out in the vicinity of Paris over a period of years on the annual development of vine mildew (*Plasmopara viticola*), *Oidium* (*Uncinula necator*), and anthracnose (*Elsinoe vitis*) [*E. ampelina*: *R.A.M.*, xxii, p. 419] and on vine resistance to early frosts in autumn led to the following conclusions. Locally, mildew generally develops later than in the south of France, occurring most frequently in the middle of summer, generally during the period when the minimum temperatures are often over 10° C. Amount of rainfall having only an uncertain relationship with the conditions of humidity that favour infection, rainfall measurements should be replaced by measurements of atmospheric humidity for practical purposes. Experimental evidence showed that five spray applications made at dates shown by annual statistics to be most suitable should usually suffice if supplemented by further treatments applied during the periods of active growth of the vine, and by two applications of copper dust to protect the bunches. These treatments are, however, effective only if carried out with care.

In 1938 and 1939, *U. necator* was exceptionally severe at Versailles. At first it was confined to certain branches which were completely infected, though the surrounding ones remained healthy, but towards midsummer it became generalized and completely destroyed the fruit clusters. On the Carignane variety it caused three-quarters of the leaves to fall prematurely. Spraying with Bordeaux mixture afforded marked protection to the leaves, but appeared to have no effect at all on the fruit, possibly owing to lack of adhesiveness.

Anthracnose has persisted for five or six years at Versailles on the hybrid Seibel 5813 growing under shade, though this and other varieties have remained unaffected in sunny situations.

ARNAUD (G.). **La Vigne et l'électricité atmosphérique.** [The Vine and atmospheric electricity.]—*Ann. Epiphyt.*, N.S., vii, 1, pp. 55–63, 2 pl., 2 diags., 1 graph, 1941. [Received September, 1945.]

During July, 1940, the author examined vines growing in the vicinity of Bordeaux which had been affected by electrical discharges from the atmosphere during stormy conditions without rain in the preceding month [cf. *R.A.M.*, xiii, p. 423; xviii, p. 152]. Most of the damage occurred on the principal branch of the year, and consisted of a superficial browning of the parts between the internodes, the tissue of the nodes in most cases remaining unaffected. Within the branch, the discoloration usually affected the medulla, where an empty space was sometimes left, following contraction of the tissues against the inner surface of the wood. The young lateral shoots generally remained healthy at first, though sometimes

they showed a browning of the medulla at their base; in some instances these shoots wilted, as water was unable to ascend into the lower, and most affected, parts. In general, it may be said that the effect became clearly defined as soon as the action causing it had taken place, only badly affected organs took several weeks to succumb, while those less severely affected became temporarily arrested and later resumed growth.

ANNAND (P. N.). **Report of the Chief of the Bureau of Entomology and Plant Quarantine, Agricultural Research Administration, 1944.**—56 pp., 1945.

In this report it is stated that phony peach disease [cf. *R.A.M.*, xvii, p. 125; xxi, p. 378] has apparently been eradicated from six entire States, and reduced by 98 per cent. in eight others, since 1936. Continued progress in control has permitted the lifting of quarantine regulations in 18 additional counties in five States; no spread to new counties was detected. It has also been possible to remove a county in Utah from the peach mosaic-regulated area [ibid., xxii, p. 381]; the number of nurseries ineligible for certification has decreased annually, and no spread of the disease from nursery stock shipments has been recorded for six years.

No trace of citrus canker [*Xanthomonas citri*] can now be found in Texas, and Federal participation in inspection has been withdrawn. Federal financing of eradication of Dutch elm disease [*Ceratostomella ulmi*: ibid., xxiv, p. 211] has also been discontinued, States and property-owners being now responsible for removing infected trees. Emphasis is now transferred from eradication of known sources of infection to the control by quarantine of spread.

Progress in the campaign against white pine blister rust [*Cronartium ribicola*: ibid., xxii, pp. 118, 119] is reviewed (p. 40), and notes are also given (pp. 44–46) on barberry eradication in relation to wheat stem rust [*Puccinia graminis*] control.

MARCHAL (E.). **Observations et recherches effectuées à la Station de Phytopathologie de l'État pendant l'année 1939–1940.** [Observations and researches carried out at the State Phytopathological Station during the year 1939–1940.]—*Bull. Inst. agron. Gembloux*, ix, 1–4, pp. 1–15, 3 figs., 1940; x, 1–4, pp. 5–10, 1941. [Flemish, German, and English summaries.]

In the first of these two reports [cf. *R.A.M.*, xix, p. 133] it is stated that during 1939 *Phytophthora erythroseptica* was recorded on potatoes in Belgium for the first time. Young flax was attacked by *Verticillium dahliae*, generally during May and June; the disease affected the external tissues of the roots and the root hairs, impeding water absorption and mineral nutrition, thus causing the plants to wilt and die. The condition has probably been present for many years, but has hitherto escaped detection. Asparagus growing near Keerbergen was attacked by *Fusarium culmorum* [ibid., xviii, p. 431], also a new record for Belgium. Soil disinfection being impracticable economically, the cultivation of this crop should be abandoned for the time being in badly affected areas. The disease was most prevalent in old plantings and in those where the general treatment of the plants, particularly manuring, was not carried out satisfactorily. Apricot mosaic (peach mosaic virus) [cf. ibid., xx, p. 370] was recorded for the first time in Belgium.

In the second report (p. 7) it is stated that flax was again attacked by *V. dahliae* in 1940.

Annual Report of the Science Service, Dominion Department of Agriculture, for the year ended March 31st, 1944.—72 pp., 1944.

Owing to war conditions it has not been found possible to publish annual reports of the Science Service of the Dominion Department of Agriculture, Canada, since 1938, when a general reorganization of the Department led to its establishment

of the Science Service as a separate unit. The Plant Protection Division was added to it in 1942 [*R.A.M.*, xxii, p. 9]. This is, therefore, the first annual report published by the Science Service, and several of its many points of interest are noted below.

Although rejections on account of bacterial ring rot [*Corynebacterium sepedonicum*] in potatoes have declined, the Departments of Agriculture of Alberta, Manitoba, New Brunswick, and Ontario now undertake surveys on their commercial growing areas; and more thorough and energetic methods for eradicating the disease are being demanded. The disease has not been found in Nova Scotia, apart from one case in 1939, which was completely stamped out. A single case was reported and dealt with by the authorities in British Columbia during 1943–4. No bacterial ring rot was observed on Prince Edward Island in 1943, only one record was made in 1942 and two in 1941; infected crops were sold to a starch factory and the storages, machinery, and tools were disinfected.

Turnip seed-producers in the Maritime Provinces have agreed to supply Ontario growers with treated seed, which has successfully controlled infection by *Xanthomonas campestris*, an increasingly serious pathogen in Ontario during recent years. The three Maritime Pathological Laboratories are treating all foundation, élite, and approved swede seed planted in the Maritimes for seed production with hot water, followed by an approved dust treatment prior to distribution. All registered swede seed for sale to the trade will have approved treatment in the cleaning plants before packaging. Experiments show that swede seed treated with ceresan or thiosan suffers no loss of germinating power, and should safely overwinter in 1 lb. packets in storage ready for sale and sowing in the spring. Other kinds of treated seed remain to be tested for unimpaired survival in winter storage.

A medium compounded by S. S. Ivanoff for *Bacterium tumefaciens* and used successfully as a selective medium for *Pseudomonas medicaginis* var. *phaseolicola* by R. D. Wilson [ibid., xvii, p. 577], is suggested for planting out seeds suspected of being infected by this pathogen.

Magnesium deficiency in potatoes, hitherto insignificant, caused great loss on Prince Edward Island in 1943 and found the farmers quite unprepared for its control. Factors favouring the disorder were high soil acidity (P_H 4·5 to 5·2), heavy acid-forming non-magnesium dressings, low organic matter in the soil, and leaching and erosion of magnesium under prolonged rainfall. Timely and adequate spraying with magnesium sulphate corrected the deficiency and averted heavy crop losses.

Investigations conducted at Fredericton, New Brunswick, have shown that potatoes may suffer severely as a result of the toxic effect of aphid feeding alone, but reaction to this injury varies widely. Susceptible varieties stimulate the production of winged forms which leave the plants as injury progresses, a factor of importance in the dissemination of virus diseases.

Severe losses of carrots in Nova Scotia and continued loss in British Columbia, both due to *Alternaria radicina* [ibid., xxii, p. 340] were reported during 1942–3, seed treatment in both cases offering an encouraging method of control.

St. Catharine's Laboratory, Quebec, has experimented successfully in three orchards with powdered cyanamide as a floor treatment designed to prevent apothecial sporulation of the peach brown rot fungus [*Sclerotinia fructicola*]. Early spring cultivation is considered, however, to control this danger equally effectively. In the same orchards, wind-blown spores from adjacent peaches, plums, and cherries caused rot at harvest where there was virtual immunity from rot at blossoming.

The Ontario maize-breeding studies [ibid., xxiv, p. 363] demonstrated the superiority of the puncture method of inoculation in producing consistently satisfactory indexes of infection in stalks and ears. *Diplodia zeae* showed greater

virulence than any of the other pathogens in stalk infection, and host susceptibility was found to increase with the advance of maturity. *Fusarium moniliforme* [*Gibberella fujikuroi*] caused almost as much damage as *D. zeae*.

Nineteenth Annual Report of the Department of Scientific and Industrial Research, New Zealand, 1944–1945.—65 pp., 1945.

In this report [cf. *R.A.M.*, xxiii, p. 473] it is stated that four applications of Bordeaux mixture (3–4–50) reduced bacterial wilt of beans [*Pseudomonas medicaginis*] from 76 to 2·7 per cent., cuprox (5 per cent.) giving comparable results. Resistance trials with 137 lines comprising approximately 70 varieties showed that all white-seeded varieties are highly resistant to wilt (and to anthracnose [*Colletotrichum lindemuthianum*]), that all runner varieties are seldom seriously infected on the pods, though their foliage may be moderately affected, and that a number of varieties with coloured seed are strongly resistant, particularly Hawkesbury, Canadian Wonder, Pinto, Pink, Tennessee Greenpod, Ne Plus Ultra, and many strains of Refugee.

A further outbreak of citrus canker [*Xanthomonas citri*: ibid., xxiii, p. 294] in Tauranga necessitated the destruction of diseased trees in an area where eradication had not previously been enforced. As three years have elapsed since the last outbreak in Kerikeri, it is evident that the eradication of infected orchards gives adequate control.

In a rapid survey in North and Mid-Canterbury, wheat eye spot [*Cercosporella herpotrichoides*: ibid., xxii, p. 351] was found only at Lincoln on a few plants in a small experimental plot at the Wheat Research Institute farm.

Raspberry rust (*Phragmidium rubi-idaei*) [ibid., xx, p. 596] became widespread at Palmerston North, and was also found in commercial areas in Wellington Province; severe defoliation sometimes followed leaf infection. Tomato streak caused heavy losses in Hutt Valley, Wanganui, and Cheviot; inoculations with the pure virus showed the disease to be identical with *Lycopersicum* virus 1 of Smith, 1937 [a strain of tobacco mosaic virus]. Preliminary investigations into the cause of yellow leaf of *Phormium tenax* have begun; a brief survey of the Moutoa area, in the Manawatu, showed that incidence there ranges from a trace to 85 per cent. Some varieties appear to be more susceptible than others, but none is immune.

Commercial methods were devised for treating soils with chloropicrin [ibid., xxiv, pp. 78, 172], which controls damping-off and *Verticillium* wilt. A new product, D.D. (a mixture of dichloropropane and dichloropropylene) gave comparable results, while being cheaper and less objectionable to use.

In five years of growth, twelve new peach varieties showed almost complete immunity from leaf curl [*Taphrina deformans*]. In the Roxburgh area, peaches showing mottle leaf with defoliation and loss of vigour, and sprayed with manganese sulphate and hydrated lime in 1943–4 and again in 1944–5, now present healthy foliage and normal vigour [cf. ibid., xxii, p. 252].

Several tobacco gardens showed the presence of angular leaf spot [*Pseudomonas angulata*: ibid., xxiv, p. 475]. An examination of five lots of 200 plants in the tobacco gardens of 21 growers in Dovedale, Motueka, and Riwaka revealed a great reduction in initial mosaic as compared with the previous season, the average percentage being only 8·7, as against 37·1 in 1943–4. In a test designed to demonstrate the ease with which mosaic can be spread by workers handling tobacco plants [cf. ibid., xvii, p. 560; xix, p. 371], the controls (clean hands) showed 5 per cent. infection, plants pulled with infected hands 36 per cent. infection, those laid out with infected hands 67·5 per cent., and those pulled after thorough washing of hands, 1·5 per cent.

Annual Report of the Agricultural Experiment Station, Rio Piedras, Puerto Rico, 1942–43.—59 pp., 1944.

The following subjects of phytopathological interest are noted from this report [*R.A.M.*, xxi, p. 408]. A strain of *Fusarium moniliforme* [*Gibberella fujikuroi*] seriously attacked field maize at the Isabela Substation. Seed of a new cucumber produced by the Agricultural Experiment Station for resistance to downy mildew [*Pseudoperonospora cubensis*] has been distributed to many growers, and progress towards the production of mosaic-resistant varieties of pepper [*Capsicum*], pumpkin, and tomato is reported.

Tobacco virus 1 [tobacco mosaic virus] and the 'mirasol' pepper virus, described by Roque and Adsuar [ibid., xxi, p. 401] were found responsible for two types of tomato mosaic. Tomatoes grown on the Station grounds were seriously affected by the 'mirasol' virus, mechanical transmission of which was found to be difficult from tomato to tomato, but easy from tobacco to tomato. The virus entity became inactivated in dead plant tissues of diseased tomato and tobacco plants. During the summer of 1942, it was practically impossible to grow tomatoes in the Rio Piedras region on account of this disease.

Report of the Federal Experiment Station in Puerto Rico, 1944.—44 pp., 1945.

In the section of this report dealing with seed treatment (pp. 28–29), D. L. Stoddard, after pointing out that seed rots and pre-emergence damping-off are important diseases of the Seminole variety of soy-beans in Puerto Rico, states that valuable protection against these conditions was afforded by seed treatments with arasan, semesan, and spergon, which gave increases of 10 to 100 per cent. in the stands.

Both semesan and cuprocide caused damage to *Cinchona* seed and reduced the stand of seedlings by 90 per cent. or more, but liquid applications of both made after germination were not injurious and controlled the spread of damping-off.

D. L. Stoddard and J. R. Pérez (pp. 34–35) adduce evidence showing that the incidence of root rot of vanilla [*R.A.M.*, xix, p. 201] is greatly reduced and the vegetative and reproductive growth of the plant improved where calcium is available to the plant.

Flint (L. H.) & Moreland (C. F.). **Note on gall formation in decapitated young Bean plants.**—*Plant Physiol.*, xx, 3, pp. 453–456, 1 fig., 1945.

Greenhouse tests on decapitated young bean plants indicated that gall formation is not essentially correlated with the action of extraneous agents, such as growth substances or crown gall bacteria (*Phytomonas* [*Bacterium*] *tumefaciens*): these results are interpreted as evidence that under some conditions the action of natural wound hormones might modify or obscure the extent or nature of gall development considered as a plant response to an applied growth substance.

Brown (J. G.) & Boyle (Alice M.). **Application of penicillin to crown gall.**—*Phytopathology*, xxxv, 7, pp. 521–524, 1 fig., 1 diag., 1945.

The writers' successful experiments in the control of crown gall (*Agrobacterium* [*Bacterium*] *tumefaciens*) on *Bryophyllum* by the application of crude penicillin from *Penicillium notatum* have already been noticed from another source [*R.A.M.*, xxiv, p. 406].

Minz (G.). **Cereal diseases in Palestine.**—48 pp., 24 figs., Agricultural Research Station, Rehovot, 1945. [Hebrew.]

An account is given of the chief diseases of wheat, barley, oats, maize, sorghum, and rye found in Palestine, together with notes on their control by seed treatment.

Ling (L.). **Aecial hosts of Puccinia graminis in China.**—*Phytopathology*, xxxv, 6, pp. 417–420, 1 fig., 1945.

Thirteen Berberidaceae have been shown by herbarium examination and field observations in western Szechuan to harbour the aecidial stage of *Puccinia graminis*, viz., *Berberis acuminata*, *B. henryana*, *B. julianae*, *B. levis*, *B. pruinosa*, *B. sargentiana*, *B. silva-taroucana*, *B. virgetorum*, *B. wilsonae*, and *Mahonia* [*B.*] *fortunei*. Of these, *B. silva-taroucana*, *B. virgetorum*, and *B. fortunei* are recorded for the first time as carriers of the rust, while *B. gagnepainii*, *B. julianae*, and *B. sargentiana*, classified as immune or highly resistant in the United States [*R.A.M.*, xi, p. 628; xvii, p. 21], were heavily infected in China. Another species, *B. dielsiana*, listed as immune from *P. g. tritici* in inoculation tests in Minnesota, usually sustains heavy damage from the rust in the mountains of northern Honan, where it is widely distributed. These observations probably point to the existence in China of virulent physiologic races of *P. g. tritici*, and the introduction into that country of reputedly immune or resistant barberries should not be effected without stringent safeguards.

Peturson (B.), Newton (Margaret), & Whiteside (A. G. O.). **The effect of leaf rust on the yield and quality of Wheat.**—*Canad. J. Res.*, Sect. C, xxiii, 4, pp. 105–114, 1945.

In continuation of earlier experiments at Winnipeg on the effect of *Puccinia triticina* on the yield and quality of wheat [*R.A.M.*, xix, p. 204], field tests were conducted in 1940 with the resistant varieties Thatcher, Renown, Regent, and Apex, in 1941 with Thatcher and Renown, and in 1943 with all four. Half the plots of each variety were kept as free from infection as possible by dusting with kolodust, while the remainder were inoculated with a mixture of some 30 different physiologic races of the rust.

The results showed that heavy infection materially reduced the yield of seed in all four varieties. Light infection depressed the yield of seed, but proportionately less than did heavy infection. Reductions in bushel and kernel weights were substantial from heavy infection and measurable from even moderately light infection. The disease generally caused slight reductions in the number of kernels per head. Analysis of variance results showed that, with the exception of the difference in bushel weights of Regent in 1940 and the differences of yield and bushel weight of Renown in 1941, the reductions in yield, in bushel weight, and in kernel weight were statistically significant. None of the differences in number of kernels per head between comparable rusted and control plants were, however, significant. Leaf rust increased the percentage of protein of the seed in 1940, and decreased it in the other years. The results of milling and baking tests showed that both in 1940 and 1943, infection slightly reduced the flour yield of all four varieties, and increased the yellow pigment content of the flour. The milling and baking quality of the grain was not, however, adversely affected in any other respect, the flour milled from the rusted samples (except Renown in 1940) being even slightly superior in baking strength to that from the non-rusted samples.

In the greenhouse experiments in 1940 and 1941, Thatcher wheat in pots was inoculated with race 76 of the rust at the seven-leaf stage, the heading stage, and the flowering stage, one group being left uninoculated as controls.

It was found that infection significantly reduced the number of kernels per head, the kernel weight, and the yield of seed in all the infected groups in both experiments. Straw weight, except in the group infected at flowering in 1940, was also significantly reduced. The greatest reductions in yield and in number of kernels occurred in the plants infected in the seven-leaf stage, and the lowest in those infected in the flowering stage. The plants infected at the seven-leaf stage produced plumper and heavier kernels than those inoculated at flowering, but many

fewer kernels per plant. The tillers in the groups infected at the seven-leaf and heading stages in 1940, and those in all three infected groups in 1941 were significantly reduced by the rust, mortality in the plants infected earliest being particularly heavy.

Leaf rust significantly reduced the height of the plants infected at the seven-leaf stage in both tests. Most of the loss of yield in the plants infected at the seven-leaf stage was due to reduction in the number of kernels, only a small proportion of it being due to a reduction in kernel weight. On the other hand, in plants infected at flowering, loss in yield was mainly due to reduction in kernel weight, and only secondarily to reduction in the number of kernels. Infection generally increased the protein content of the leaves, and to a less extent of the seed straw.

Bremer (H.). **Bunt of Wheat in Turkey.** *Zir. Derg.*, iv, 44–45. pp. 124–130, 1943. [Turkish, with English summary. Received September, 1945.]

Effective control of wheat bunt [*Tilletia caries* and *T. foetida*] would raise production in Turkey [*R.A.M.*, xix, p. 464] by 15 to 20 per cent., and recommendations for the accomplishment of this object by standard methods of seed treatment are given. However, the development of resistant wheats from indigenous varieties, some of which show promising resistance, would render such measures superfluous.

Johnson (F.). **The effect of chemical soil treatments on the development of Wheat mosaic.**—*Ohio J. Sci.*, xlv, 4, pp. 125–126, 1945.

Calcium cyanide (granulated) at 1, 3, and 5 gm., carbon disulphide at 2, 5, and 10 c.c., chloropicrin, ethyl chloride, ethylene dichloride, and methyl bromide at 0·3, 1, and 3 c.c., commonly used as insecticides and nematocides, were tested by the author as agencies for controlling wheat mosaic [*R.A.M.*, xxiv, p. 407] by adding them to screened soil at a temperature of 16° C., placed in air-tight metal containers and then transferred after sealing to a greenhouse, where the temperature was approximately 27°. The infested treated soil was removed from the metal containers, in which the chemicals had been placed half-way between the bottom and the surface of the soil, aerated, transferred to No. 10 tin cans perforated to allow of drainage, and sown with Purdue No. 1 wheat, a variety highly susceptible to mosaic. After sowing, the cans were placed out of doors over winter in soil trenches 8 in. deep.

Three examinations were made up to the time that the wheat had entered the boot stage and calcium cyanide was shown to give complete control at 5 gm., but none at 1 gm.; carbon disulphide at each concentration tested gave complete control, and chloropicrin and methyl bromide likewise. Ethyl chloride was ineffective and ethylene dichloride gave only incomplete control at the higher concentrations. In another experiment, fumes of naphthalene and rotenone dust, when mixed with the soil, gave complete control of wheat mosaic. It is thought that the chemicals acted on the vector rather than on the virus.

Yu (T. F.) & Fang (C. T.). **A preliminary report on further studies of physiologic specialization in Ustilago hordei.**—*Phytopathology*, xxxv, 7, pp. 517–520, 1945.

Most of the 84 collections of covered smut (*Ustilago hordei*), obtained chiefly from south-western China [*R.A.M.*, xxii, p. 427], were inoculated from 1941 to 1943 into 14 barley varieties, of which four were selected as differential hosts, namely, Nanking Nos. 368 and 373, Himalaya (C.I. 1312), and Excelsior (C.I. 1248). On this basis nine physiologic races of the pathogen were recognized and designated by the numbers 1 to 9. The two predominant races were 1 and 2, of

which the former is the weakest of all in pathogenicity, not only the four differential varieties, but the rest of those inoculated being resistant to it, while Himalaya and the two Nankings are likewise resistant to race 2 and Excelsior intermediate in its response.

BEVER (W. M.). **Hybridization and genetics in Ustilago hordei and U. nigra.**—*J. agric. Res.*, lxxi, 2, pp. 41–59, 4 figs., 1945.

The production of new physiologic races from interspecific and intraspecific hybridization of *Ustilago nigra* and *U. hordei* is described [*R.A.M.*, xxii, p. 351; xxiv, p. 143]. The temperature requirements for germination of the two species and of two F_2 hybrids between them were similar, the optimum ranging from 24° to 28° C. Chlamydospores of *U. hordei* showed up to 50 per cent. germination at 36°, whereas those of *U. nigra* failed to germinate at that temperature. *U. hordei* hybridized readily with *U. nigra* as shown by sporidial fusion in culture and hybrid chlamydospore formation in the host. Interspecific and back-crosses failed to show simple Mendelian inheritance of chlamydospore markings, pathogenicity, and type of smutted head. The smutted heads produced from a monosporidial interspecific cross ranged in the F_1 from those closely resembling *U. nigra* to the *U. hordei* type. Whereas back-crosses of the interspecific hybrids on the *U. hordei* parent tended to yield heads of the *U. hordei* type, most of the interspecific hybrids on the *U. nigra* parent gave rise to all types of heads. In the F_2 of the interspecific hybrid some correlation was observed between chlamydospore markings and type of smutted head, the *U. nigra* type usually having echinulate spores and those of the *U. hordei* type smooth or echinulate with roughly equal frequency.

The interspecific hybrid *U. hordei* H6 × *U. nigra* N6 proved pathogenic to the three test varieties Nepal, Lion, and Himalaya, although *U. hordei* H6 attacked only Lion. Similarly, the intraspecific hybrid *U. nigra* N4 × *U. nigra* N6 attacked all three varieties, whereas *U. nigra* N4 attacked only Lion. Hence both hybrids differed from their parents in degree of pathogenicity. In the F_2 both hybrids tended to combine pathogenic characters rather like the F_1, except that the F_2 of the interspecific hybrid produced more smutted heads on Himalaya than either of its parents. It is apparent that new physiologic races may result from hybridization between the two species studied. The fact that some heads smutted by the interspecific hybrid contained only smooth chlamydospores, while others showed only echinulate ones, suggests that the inbred races of *U. hordei* and *U. nigra* used in the experiments were phenotypically pure.

ARNY (D. C.). **Physiologic specialization in Helminthosporium gramineum Rabh.**—*Phytopathology*, xxxv, 7, pp. 571–572, 1945.

Recently a culture of barley stripe (*Helminthosporium gramineum*) pathogenic to the Atlas variety, which is normally resistant to the disease in Wisconsin [*R.A.M.*, xxiii, p. 479] though susceptible in California [ibid., xxii, p. 473], was isolated from a spontaneously infected plant at the Agricultural Experiment Station of the former State. It was designated G.H.A 2 and tested on Oderbrucker (C.I. 4666), Colsess IV (C.I. 5979), and three lots of Atlas, of which one (Wisconsin) had been included in the State breeding programme for a number of years, the second (California) was supplied by C. A. Suneson, and the third (C.I. 4118) was grown by R. G. Shands at the Wisconsin Agricultural Experiment Station in 1942. Two other cultures of *H. gramineum*, C–1 and 45–2, both of Wisconsin origin, were used for comparative purposes.

Atlas from all three sources responded similarly to inoculation, being resistant to C–1 and 45-2 but susceptible to G.H.A 2 (average infection on Wisconsin 3, 3, and 72 per cent., respectively, on California 0, 59, and 0, and on C.I. 4118 0, 92, and 0). Oderbrucker was susceptible to C–1 and 45–2 (86 and 97 per cent.,

respectively), but highly resistant to G.H.A 2 (1 per cent.), while Colsess was severely attacked by all three (87, 100, and 80 per cent.) respectively. The new culture is thus pathogenically distinct from the other two.

Moore (M. B.), Hayes (H. K.), & Stakman (E. C.). **The field reactions of certain Oats hybrids and varieties to stem rust.**—*Phytopathology*, xxxv, 7, pp. 549–551, 1 fig., 1945.

Selected lines from crosses made from 1930 to 1933 at the Minnesota Agricultural Experiment Station between Bond oats and each of the varieties Rainbow, Iogold, Anthony, Double Cross A, and Double Cross B (Minota × White Russian) × Black Mesdag were consistently resistant to stem rust (*Puccinia graminis avenae*) until 1943, when nearly all the lines from Bond × Rainbow and Bond × Iogold bore a liberal sprinkling of large pustules besides the usual number of small ones, the former indicating the presence of races pathogenic to these varieties. The Vicland and Tama varieties also showed both types of pustules. Race 8 was exclusively isolated in the greenhouse from all the nine collections from lines or varieties with Rainbow, Iogold, or Richland parentage (Tama and Vicland from the last-named), while three out of four from Anthony or Double Cross yielded only race 2, the remaining one producing 2 and 8 in the proportions of 70 and 30 per cent., respectively.

These observations are briefly discussed in the light of recent studies on physiologic specialization in oats stem rust, including those of Stakman and Loegering [*R.A.M.*, xxiii, p. 433], demonstrating the extensive increase of race 8 in 1943.

Stanton (T. R.). **Registration of varieties and strains of Oats, XIV.**—*J. Amer. Soc. Agron.*, xxxvii, 8, pp. 643–644, 1945.

Mission (C.I. 2588), Reg. No. 104, originating from a cross between Markton and Victory oats made at Aberdeen, Idaho, in 1923, approved for registration in 1944 [cf. *R.A.M.*, xxii, p. 299], is a medium-tall, early to mid-season variety with spreading panicles, relatively stiff straw, and plump, white kernels, which has shown a high degree of resistance to the races of loose and covered smuts [*Ustilago avenae* and *U. kolleri*] occurring in Montana.

Altstatt (G. E.). **A new Corn disease in the Rio Grande Valley.**—*Plant Dis. Reptr*, xxix, 20, pp. 533–534, 1945. [Mimeographed.]

An apparently new maize disease, possibly of a virus nature and characterized by the stunted, bushy appearance of affected plants with chlorotic, green- and white-striped leaves, and pronounced broad bronze and red bands extending along the whole length of the leaf, has been engaging the attention of plant pathologists in the Rio Grande Valley, Texas. It is highly prevalent in the Weslaco-Donna area, the percentage of plants affected in commercial fields varying from 0 to 30, and well-known varieties of field, sweet, and popcorn were attacked. A further consistent symptom of the disease is the growth of an elongated ear shoot, often exhibiting as many as five nodes, from each of the lower nodes of the plant, and each shoot may produce two to three vestigial or poorly developed ears. Some plants affected may produce a normal full ear, but later ears may be incompletely developed, with poorly filled kernels. In some cases there is no emergence of silk, the tips of the husks become dry, and in sweet maize particularly, the flag leaves on the ear are much longer in proportion to the length of the ear, and usually narrower, than those on healthy ears. The angle between these leaves and the ear is also wider. There is no evidence of internal breakdown, cavities or discoloration in the stalk, though the basal tip sometimes shows necrosis. The roots are usually sound. Research is continuing with a view to determining the exact nature of this disease.

Saha (J. C.) & Bhattacharjee (S. K.). **Artificial production of ergot in the tropical plains of Bengal.**—*Nature, Lond.*, clvi, 3960, pp. 363-364, 1 fig., 1945.

In an experiment carried out at Calcutta, four small plots were sown with rye and sprayed at flowering with a conidial suspension of *C[laviceps] purpurea*, obtained by culturing pieces of ergot from a commercial source. Good infection of individual rye flowers occurred on all plots, the sclerotia maturing seven to nine weeks after the first spraying. From 1 to 33 (commonly 3 to 6) of these organs were present on each panicle, the largest measuring 3·6 by 0·4 cm. and weighing 0·248 gm. when air-dried. The sclerotia were hand-picked, and after six weeks' storage at room temperature (20° to 25° C.) were sent in glass bottles for assay. The report received stated that the ergots were of good quality, with total alkaloid and ergometrine contents of about 320 and 72 mg. per 100 gm., respectively [cf. *R.A.M.*, xxiii, p. 382]. This result demonstrates that very good ergot can easily be produced artificially in the plains of the tropics.

Johann (Helen) & Dickson (A. D.). **A soluble substance in Cornstalks that retards growth of Diplodia zeae in culture.**—*J. agric. Res.*, lxxi, 3, pp. 89–110, 14 figs., 1945.

Maize plants are usually resistant to stalk rot caused by *Diplodia zeae* during the vegetative period of their growth. Susceptibility increases after pollination and the disease is seldom noticeable until the milk stage has been reached and the kernels have become dented. These facts have led the authors to seek evidence of physiological resistance to stalk rot in an ether- or sap-soluble substance in the stalks of yellow dent maize, and they present in this paper the results of experiments conducted during the years 1935 to 1940.

The ether extracts of 11 inbred lines and 5 hybrids collected before or shortly after pollination usually delayed the development of the fungus to a greater degree than extracts drawn from stalks at a later period of growth. Some lines retained more of the growth-retarding substance throughout the maturation period than others, suggesting a physiological type of resistance not necessarily associated with strength of stalk or morphological characters.

The growth of *Gibberella zeae* and *Nigrospora sphaerica* was similarly inhibited to some extent by the ether extract, but *G. fujikuroi* showed little susceptibility to it. The growth-retarding substance was stable to heat and slightly soluble in water, and removal of the ears did not increase this property, or clipping of the leaves lower it.

Expressed juice of the stalks contained a growth-retarding substance that showed seasonal change and differed in the lines tested as did the ether extracts. After the time of pollination, the capacity to retard growth grew less, noticeably in the case of susceptible tissue. The chemical nature of the substance is not known, but it is suggested that the differences exhibited in the physiological resistance of the various lines may be caused by differences in timing and in the speed with which lines pass through the various stages of their development, rather than by fundamental differences in the general trend of the curve of susceptibility.

Standen (J. H.). **Nigrospora oryzae (B. and Br.) Petch on Maize.**—*Phytopathology*, xxxv, 7, pp. 552–564, 2 figs., 1 graph, 1945.

The following conclusions are drawn from the author's investigations at the Iowa Agricultural Experiment Station on the ear rot of maize caused by *Nigrospora oryzae* [*R.A.M.*, xxiii, p. 339]. Poorly-matured tissues were more susceptible to infection than well-matured ones. Single crosses (236 in all) were separated on the basis of visual inspection into three groups, i.e., poorly- and well-matured and prematurely dying, numbering 66, 101, and 69, respectively, the incidence of infection in which was 6·8, 4·9, and 19·7 per cent., respectively. Lodging, stalk and

root rot, and early frosts were all contributory factors in an increased incidence of ear infection, which was also high in secondary ears and axillary shoots on account of their poor maturity. Such material probably affords a principal source of inoculum for the following season.

The relation between the P_H of the cob and the amount of infection, previously observed by Reddy [ibid., xiii, p. 299], depended on the lowering of the cob P_H as the ear matured. In cobs maturing poorly or still immature at the onset of frost, the P_H remained high. Late-maturing inbreds were heavily infected in years of early frost and remained healthy when frost was delayed. Green tissues did not contract the disease.

In many ears the fungus failed to spread beyond the incipient stage of infection, its further growth being apparently conditioned by moisture, temperature, and cob maturity. The retting of cob tissues, a characteristic field symptom, was produced in the laboratory on insufficiently matured samples only. Chaffiness of the ears, another feature of the disease, is probably also a concomitant of the incomplete maturity predisposing to attack by *N. oryzae*.

Moerdyk (J. L.). **Division of Agriculture.**—*Rep. vet. agric. Dep. Swaziland, 1944*, pp. 13–19, [1945. Mimeographed.]

Information received from H. Hutchinson stated that late plantings of maize at the White Umbuluzi farm, Swaziland, were severely attacked by streak [*R.A.M.*, xxiii, p. 296], and yielded only about two bags per acre, as against a general average of six. Seed of a resistant hybrid of the Peruvian and Hickory King varieties from Barberton gave a crop notably free from the disease, even though planting was not until March. The seed is being multiplied for distribution.

Arentsen (S.). **Estudio de la susceptibilidad presentada por diversas especies y variedades de Citrus al ataque de Phytophthora citrophthora (Sm. & Sm.) Leon.** [Study of the susceptibility to attack by *Phytophthora citrophthora* (Sm. & Sm.) Leon. of certain species and varieties of Citrus.]—*Agric. tec. Chile* (formerly *Bol. Sanid. veg. Chile*), ii, 1, pp. 54–58, 1945.

Inoculum from a pure culture of *Phytophthora citrophthora* [*R.A.M.*, xxii, p. 477], isolated by Fawcett and Bitancourt in 1937 from fallen fruit of bitter oranges found in Misiones Province, Argentina, was introduced into the bark of citrus trees through holes made by a blunt instrument. The resultant lesions varied considerably in size among the different species, the areas affected on lemon, Persian lime, citrangequat [Willits citrange (= *Poncirus trifoliata* × common orange) × oval kumquat (*Fortunella margarita*)], Satsuma orange [*Citrus nobilis* var. *unshiu*], and grapefruit being 183·3, 59, 56·5, 56, and 55 (54·2 in another test) sq. cm., respectively. Variations also occurred among varieties of the same species (Villafranca, Eureka, and Ponderosa varieties of lemon) and even in individual fruits of the same variety (of which the Persian lime fruit was an outstanding example), without it being possible to determine any significant difference in the relative susceptibility of trunks and branches. In general, the results of the experiment confirmed those obtained by Klotz and Fawcett in 1930 [ibid., ix, p. 303] and Fawcett and Bitancourt [ibid., xx, p. 400], and demonstrate the susceptibility of the lemon to *P. citrophthora*.

Cass Smith (W. P.). **A note on the occurrence of Sclerotinia twig blight of Oranges.** —*J. Dep. agric. W. Aust.*, xxii, 1, pp. 77–80, 2 figs., 1945.

The author believes that this is the first occasion on which *Sclerotinia sclerotiorum* has been noted on the woody parts of citrus [*R.A.M.*, xxiii, p. 481] in Western Australia, although the fungus has a wide host range, and is frequently active in cold weather, in this and other parts of the Commonwealth [ibid., xxi, p. 440]. Infection took place at injuries due to frost on the twigs of orange trees in an

orchard at Brookhampton, and the symptoms exhibited were similar to those described as characteristic by Fawcett, including the shredded fibrous condition, which distinguishes it from a similar trouble caused by *Botrytis cinerea*. The source of the infection is thought to be the orchard cover crop in view of the frequency with which winter cover crops are attacked by this pathogen. Excision and burning of infected twigs and the deep burial of the orchard cover crop, if also infected, is recommended. Spraying with Bordeaux mixture prior to the winter rains may give some protection.

Fawcett (H. S.). **Chemical treatment for scaly bark of Citrus.**—*Calif. Citrogr.*, xxx, 11, p. 340, 1 fig., 1945.

The author describes a chemical method of treating psorosis bark lesions early so as to prolong the commercial usefulness of citrus trees [*R.A.M.*, xxiii, p. 340]. One per cent. solution of dinitro-ortho-cyclohexylphenol in paraffin is applied with a brush to the lesion itself, and to the bark surface 6 in. above and below and 3 in. on either side. Before applying the solution, loose scales should be rubbed off with a wire brush or heavy leather glove, care being taken not to injure the live bark. Every precaution must be taken not to let the liquid run down the trunk, as it may cause the death of the bark right through to the wood. Thus, no part of the bark should receive more solution than is needed to wet it from one stroke of the brush, and brushes with very coarse bristles should not be used, as the liquid is likely to run too rapidly from them. Properly applied, dinitro-ortho-cyclohexylphenol solution penetrates and kills only the lesioned, outer bark and the cambium is able to build a new bark. The solution should be applied preferably during the months of April, May, and June, and September and October should be avoided. Each tree in an orchard should be inspected once a year in order to discover lesions as soon as they occur. Treatment at a later stage when the top has begun to deteriorate is useless.

The solution, known as DN–75, is prepared and distributed by the Dow Chemical Company.

Wallace (J. M.). **Technique for hastening foliage symptoms of psorosis of Citrus.** - *Phytopathology*, xxxv, 7, pp. 535–541, 2 figs., 1945.

At the Citrus Experiment Station, Riverside, California, the inoculation of sweet orange (mostly Homosassa) seedlings with a basal trunk diameter as small as 5 mm. has greatly expedited the development of the foliar symptoms of psorosis [*R.A.M.*, xxiii, p. 61]. Small bark patches from diseased trees are placed against the cambium below strips of bark peeled back on the trees to be inoculated. Callus is rapidly formed and the virus transmitted. The small stock trees are topped to within 2 or 3 in. of the bark patch.

Symptoms either of the severe-shock or young-leaf stippling and blotching type usually appear on the growth arising from the upper axillary buds after topping. Inoculated trees reacting by severe-shock symptoms during the first flush of growth commonly show only the typical young-leaf features of psorosis in the later flushes. If the bark patches used for inoculum are taken directly from the lesion areas, the infected stocks develop bark and mature-leaf symptoms early, often within two months, and undergo a permanent set-back to growth.

An inoculation procedure lessening the time required for symptom development in psorosis-affected orange trees is of value in the acceleration of transmission studies, while other uses are (1) as an adjunct to, and possibly after further development as a substitute for, the regular inspection of trees proposed as bud-wood parents and the field tests on their progeny; and (2) as a means for the positive substantiation of the presence of psorosis in suspected trees yielding no reliable evidence of infection in the field.

Bhat (S. S.). **The die-back disease of Citrus trees.**—*Indian Fmg*, vi, 6, pp. 250–253, 1945.

In this paper the washing down of surface soil by irrigation and natural precipitation to subsoil layers, where it packs hard and prevents drainage, is seen as a possible cause of the malnutrition of citrus trees and consequent onset of die-back disease in the Indo-Ganges and Gujarat Valleys [*R.A.M.*, viii, p. 442], where the flat riparian countryside also impedes lateral drainage, although the soil itself is favourable to citrus cultivation. Other causes of citrus malnutrition appear to be associated with the physical and chemical composition of soils, and the author commends to Indian plant pathologists the methods of soil deficiency diagnosis developed at the East Malling Research Station. Manuring of citrus plantations is backward in India. Mosambi and other citrus trees in the Punjab and Gujarat have responded well to spraying by zinc and iron sulphates. Budding delicate citrus varieties on resistant jamburi stocks and rigorous selection of root stock seedlings are also essential measures of die-back control. Plantations should be made on carefully selected soils of reasonable elevation, and salty and 'kalar' patches and soils with high lime content should be avoided.

Rudolph (B. A.) & Harrison (G. J.). **The invasion of the internal structure of Cotton seed by certain Fusaria.**—*Phytopathology*, xxxv, 7, pp. 542–548, 1945.

In the course of the writers' studies in California on the role of cotton seed in the dissemination of *Verticillium albo-atrum* [*R.A.M.*, xxiv, p. 99], several species of *Fusarium* were isolated from apparently healthy seed, which must have harboured the species internally since delinting with acid precluded the possibility of survival on the exterior. In 1935 8·19 per cent. of the placentae in bolls from plants infected with *Verticillium* wilt yielded *Fusarium* and in 1936 7·37 per cent., though the plants showed no sign of disease except that they were more or less stunted. Seed from bolls with receptacles yielding *Fusarium* in culture was delinted and planted in the field on soil not known to have borne cotton crops before. Isolations from the steles of such plants showed 25 per cent. infection with *Fusarium* as against 19·81 per cent. for plants raised from so-called healthy seed, which evidently shows that *Fusarium* was present in much of that seed when planted.

The *Fusarium* spp. reach the seed by way of the xylem, which they presumably occupy as saprophytes, since the plants display no pathological symptoms of any kind. The species so far identified are *F. moniliforme* [*Gibberella fujikuroi*], *F. oxysporum*, and *F. scirpi*, of which the last-named is capable of inducing a soft, brown rot of immature seeds.

Vasudeva (R. S.). **Studies on the root-rot diseases of Cotton in the Punjab. XIII. Leaf temperatures of healthy and root-rot affected plants.**—*Indian J. agric. Sci.*, xiv, 6, pp. 385–388, 1944.

After stating that root rot [*Corticium solani* and *Macrophomina phaseoli*: *R.A.M.*, xxiii, p. 484] is the most serious disease of cotton in the Punjab, where it causes heavy damage every year, the author describes experiments in which the leaf temperatures of healthy and affected plants were recorded by the thermo-electric method. The results obtained [which are tabulated] showed that affected plants remain considerably warmer than healthy ones. For example, the temperatures of the inner leaf tissues of healthy and diseased plants were (in one of six tests) 30·62° and 31·09° C., respectively at 6.30 a.m., 32·95° and 36·10° at 9 a.m., 37·81 and 39·28° at noon, and 38·18 and 38·83 at 5 p.m.

Preliminary work also showed that the transpiration rate is much higher in healthy than in affected plants. The increase in temperature of diseased plants is probably due to defective root absorption.

PRASAD (N.). **Studies on the root-rot of Cotton in Sind. II. Relation of root-rot of Cotton with root-rot of other crops.**—*Indian J. agric. Sci.*, xiv, 6, pp. 388–391, 1944.

In Sind, cotton root rot is caused by *Rhizoctonia bataticola* [*Macrophomina phaseoli*] and *Fusarium caeruleum* [*R.A.M.*, xxiv, p. 228 and cf. preceding abstract], singly or together. Other crops attacked by root rot include castor (*Ricinus communis*), sesame (*Sesamum orientale*), sunn-hemp (*Crotalaria juncea*), and guar (*Cyamopsis psoraloides*), and experiments were accordingly carried out to determine the cause of the disease in these hosts.

Affected roots of castor, sunn-hemp, sesame, and guar were collected at different times from different places in Sind, and isolations made from them at regular intervals. The isolates that most frequently appeared were *Fusarium* and *Rhizoctonia*. Cultures of *Rhizoctonia* from these four hosts and cotton were used to inoculate seedlings of all these, and were found to be pathogenic. Isolations were made from the inoculated plants and the cultures obtained were found in almost all cases to be identical with the cultures used for the inoculations. It is therefore evident that the rot in all these crops is caused by *Rhizoctonia* and that the fungus isolated from the rotted roots is pathologically the same in all cases. Similar experiments were carried out with the *Fusarium*, with the same results. The organisms were identified as *R. bataticola* [*M. phaseoli*] and *F. caeruleum*, respectively. The latter showed aerial mycelium and bluish-green sporodochia in all cultures while sclerotia were absent; only in the isolate from sesame was there a concentration of spores on the surface resembling pionnotes. Terminal chlamydospores were produced in abundance by each isolate on practically every medium and intercalary chlamydospores on potato and steamed rice. The conidial measurements on 2 per cent. potato dextrose agar were as follows: 0-septate 6 to 15 by 3 to 6 μ; 1-septate 9 to 20 by 3 to 6 μ; 2-septate 13 to 33 by 3 to 7 μ; and 3-septate 25 to 40 by 3 to 7 μ.

HEIM (R.). **Études descriptives et expérimentales sur les Agarics termitophiles d'Afrique tropicale.** [Descriptive and experimental studies on the termitophile Agarics of tropical Africa.]—*Mém. Acad. Sci., Paris*, lxiv, 74 pp., 10 pl. (1 col.), 1941. [Received September, 1945.]

This is a full description and critical discussion, in the light of relevant contemporary literature, of the author's studies on the Agaricaceae of tropical African termites' nests [*R.A.M.*, xxii, p. 333 and next abstracts], which he regards as constituting an entirely distinct section of the family in respect of their physiognomic, anatomical, cytological, biological, cultural, and biochemical characters. To accommodate these fungi a new genus, *Termitomyces* [with a diagnosis in French only], is erected, of which one group of three forms, including a new species, *T. mammiformis*, is comprised in the present memoir.

HEIM (R.). **Nouvelles études descriptives sur les Agarics termitophiles d'Afrique tropicale.** [New descriptive studies on the termitophile Agarics of tropical Africa.]—*Arch. Mus. Hist. nat. Paris*, Sér. 6, xviii, pp. 107–166, 4 pl. (1 col.), 19 figs., 1942. [Received September, 1945.]

Included in this further instalment of the author's investigations on the Agaricaceae of tropical African termites' nests [see preceding and next abstracts] are six species of *Termitomyces*, two newly described and two new transfers to that genus. A dichotomous key to the species and varieties of *Termitomyces* and three other synoptical tables are given, besides notes on *Marasmius pahouinensis* de Seynes and two chromosporous species of *Lepiota* saprophytically associated with termites' nests. Latin diagnoses of the new genus and species are given.

Heim (R.). **Les champignons des termitières. Nouveaux aspects d'un problème de biologie et de systématique générales.** [The fungi of termites' nests. New aspects of a problem of general biology and taxonomy.]—*Rev. sci., Paris*, lxxx, 2, pp. 69–86, 22 figs., 1942. [Received September, 1945.]

This is a summary of the essential results of the author's studies on the biology and taxonomy of the *Termitomyces* associated with termites' nests in tropical Africa [see preceding abstracts].

Couch (J. N.). **Revision of the genus Coelomomyces, parasitic in insect larvae.**—*J. Elisha Mitchell sci. Soc.*, lxi, 1–2, pp. 124–136, 2 pl., 1945.

After an historical review of the genus *Coelomomyces* [*R.A.M.*, xxiii, p. 16], with a résumé of the species hitherto known, the author describes five new species from Georgia, this being the first report of this genus from the Western Hemisphere. The new species are *C. dodgei* on *Anopheles crucians* and other species of *Anopheles*, *C. psorophorae* on *Psorophora ciliata*, *C. quadrangulata* on *Anopheles* spp., *C. pentangulata* on *Culex erraticus*, and *C. uranotaeniae* on *Uranotaenia sapphirina*. The author's observations confirmed the view that the mycelium lacks cell walls. The resting sporangia, their dehiscence, and the structure of the zoospore suggest a relationship with the Blastocladiales, but because of the naked mycelial threads and the obligate parasitism in insect larvae, the author places these fungi in a new family, the Coelomomycetaceae, in the Blastocladiales.

Karling (J. S.). **Brazilian Chytrids. VI. Rhopalophlyctis and Chytriomyces, two new chitinophyllic operculate genera.**—*Amer. J. Bot.*, xxxii, 7, pp. 362–369, 61 figs., 1945.

Pursuing his studies of the Chytrids of Brazil [*R.A.M.*, xxiv, p. 313], the author reports two new monocentric operculate genera, *Rhopalophlyctis* and *Chytriomyces*, and three new species, *R. sarcoptoides*, *C. aureus*, and *C. hyalinus*, first found on skeletons of unidentified insects in fresh water in the Amazon Valley and later collected in abundance on the exuviae of mayflies in Connecticut, New York, and Virginia.

De Salles (J. F.) & Hathaway (C. R.). **Nota sôbre a infestação de Musco domestica Linneu, 1758, por um Ficomiceto do gênero Empusa.** [Note on an infestation of *Musca domestica* Linnaeus, 1758, by a Phycomycete of the genus *Empusa*.]—*Mem. Inst. Osw. Cruz*, xli, 1, pp. 95–99, 4 figs., 1944. [English summary.]

The authors record an outbreak of disease among house flies in Rio de Janeiro, Brazil, caused by *Empusa muscae* [*R.A.M.*, xxi, p. 45], with brief notes on the fungus.

Arny (A. C.). **Registration of improved Flax varieties, III.**—*J. Amer. Soc. Agron.*, xxxvii, 8, pp. 646–648, 1945.

Three blue-flowered varieties of flax, Arrow (C.I. 1070), Renew (C.I. 839), and Koto (C.I. 842), Registered Nos. 5, 6, and 7, respectively, were approved for registration in 1944 [cf. *R.A.M.*, xxiii, p. 63]. Arrow, a selection from a Bison × Renew cross made at Bozeman, Montana, in 1938, has been found to be immune from the North American races of rust (*Melampsora lini*) and equally resistant with the Bison parent to wilt (*Fusarium lini*) and 'pasmo' (*Sphaerella linorum*). It is recommended for seed production in Montana.

Renew is a selection from the cross Newland × (Reserve × Monje) made at Mandan, North Dakota, in 1930. It is immune from rust, moderately resistant to wilt, and susceptible to 'pasmo'. It is recommended for seed production in North Dakota and Montana.

Koto, another North Dakota selection from the cross (Reserve × Monje) × Bison, is susceptible to rust and similar in wilt and 'pasmo' reaction to the Bison parent. It is on the list of recommended varieties for Minnesota and the Dakotas.

Millikan (C. R.). **Wilt disease of Flax.**—*J. Dep. Agric. Vict.*, xliii, 7, pp. 305–313, 9 figs.; 8, pp. 354–361, 5 figs., 1945.

Successive outbreaks of flax wilt (*Fusarium lini*) have occurred in Victoria in recent years. The disease in the past has often been attributed to over-cropping [*R.A.M.*, viii, p. 795], and the severe outbreaks in the Drouin district during 1941, 1943, and 1944 are stated to have occurred on overcropped land. Flax sickness has been attributed to various fungi other than *F. lini*, but it is clear that this species is the primary cause of that disease also [ibid., xx, p. 18]. In countries like Victoria, where the disease appears very late in the season, the only symptom may be a premature ripening. The critical temperatures for the development of wilt are closely parallel with those for the causal organism. Greenhouse experiments show that susceptible plants growing in naturally infected soil may escape the disease if the soil temperature is 55° F. or below. When transferred to a tank with the temperature maintained at 75°, severe infection developed in two to three weeks, 100 per cent. of the plants being infected in the case of the Concurrent variety and 50 in that of Bison.

In field experiments from 1942 to 1944 inclusive, the soil temperatures were very favourable for wilt infection in January to March and from October to December, and high percentages of infected plants occurred in sowings during these months. From May to September (most of the flax in Victoria is sown in late May or June), temperatures were below the minimum for infection and the plants showed no sign of wilt until the middle of October, after which they developed severe infection. The occurrence of wilt, therefore, is solely dependent on the prevailing soil temperature and the age of the plant has little effect on the final severity.

Flax varieties to be tested for resistance were grown at a temperature of 75° in Burnley temperature tanks in 'wilty' Drouin soil, the infection of which was considerably increased by a preliminary sowing of the susceptible Concurrent variety. In these circumstances up to 100 per cent. wilt infection was obtained within 20 days of germination. The surface of the pots was divided into two or four equal sections, and Concurrent seed sown in one section for control purposes. Where the soil had not been built up by a preliminary sowing of Concurrent, many varieties included in the susceptible class developed less wilt, but resistance tended to break down after several sowings in 'wilty' soil. This may have been due to depletion of plant nutrients in the soil, concerning the influence of which in encouraging wilt disease there is little information. The old soil was then replaced after two tests only by fresh, 'wilty', steam-sterilized soil, which was inoculated with pure cultures of *F. lini* and the inoculum built up by one or two sowings of Concurrent. As a result, the percentage of infection recorded was often higher than when the same varieties were grown in naturally infected soil. Less than 40 per cent. infection was shown by Argentine 868, Newland × (19 × 112) C.I. 839, Ottawa 770B × Buda C.I. 1073, Saginaw × Bombay C.I. 671, and Russian × Argentine C.I. 896. The last-named, which is also immune from *Melampsora lini* [ibid., xxiii, p. 437], has been selected as the foundation stock for a flax-breeding policy designed to produce wilt- and rust-resistant varieties suitable for conditions in Victoria. Russian × Argentine C.I. 896, tested for wilt reaction with Concurrent and Liral Crown varieties, in naturally infected soil in the field in a normal winter sowing, was free from infection 150 days after germination, whereas Concurrent showed 99·8 and Liral Crown 100 per cent.

In experiments on the selection of wilt-resistant strains of flax, Liral Crown, Liral Prince, Royal, and J.W.S. selections all exhibited a superior wilt reaction to the parent plant in temperature-tank tests, particularly the Royal selection. The Liral Crown and Liral Prince selections also showed high resistance in the field, the former in both winter and summer sowings and the latter in a winter sowing.

A Victoria strain (D1) of *F. lini* has been compared with two strains from New Zealand and one from Minnesota in temperature-tank tests and found to be distinct. The need for testing new flax varieties to as many races of *F. lini* as possible before release is therefore clearly demonstrated.

White (N. H.). **The relation of Polyspora lini Lafferty and Pullularia pullulans (de Bary) Berkh. to Flax browning.**—*J. Coun. sci. industr. Res. Aust.*, xviii, 2, pp. 141–149, 1945.

During 1942 and 1943 both the author and H. R. Angell observed browning of flax crops in Tasmania, though no stem-break was present. From affected material the author isolated *Pullularia pullulans*, which closely resembled isolates of *Polyspora lini* [*R.A.M.*, xxi, p. 334] from other countries. Platings from diseased stems showed five dominant fungi, viz., *Pullularia*, *Fusarium*, *Cladosporium*, *Alternaria*, and *Phoma*. Flax seed of different origins, including crops affected with browning, was tested for germination and for seed-borne organisms by the Ulster method. Germination in pots was about the same for all sources. No acervuli or other structures resembling those described by Lafferty were found on any of the tissues or seeds examined. The only fungus found on the diseased flax that resembled *Polyspora lini* was *Pullularia pullulans*, the essential points of resemblance being the production of slimy radula spores with a yeast-like budding process.

When two cultures of *Pullularia pullulans* (P1 and P2) from Canberra and Tasmania and four of *Polyspora lini* (P5, P6, P8, and P9) from two places in Victoria, New Zealand, and Ireland were compared, it was not found possible satisfactorily to distinguish them on a basis of microscopic structure, cultural, serological, and physiological characters, capacity to ret flax [cf. ibid., xxi, p. 370], or pathogenicity to flax. Two (P8 and P9) were distinguishable from P1, P2, P5, and P6 on a basis of sprout-cell size and shape and of the waxy-pink colour developed on potato slopes as against the dirty buff shown by the last four, but P5 and P6, which had been identified as *P. lini*, had characters that corresponded with P1 and P2, which were identified as *Pullularia pullulans*. Again, P8 and P9, though possessing sprout-cells of the same shape, differed significantly in sprout-cell size. The only basis for differentiation would seem to be the shape of the sprout-cells and the thallus colour. On this basis, P8 and P9 (both *Polyspora lini*) would be one group and the other four (two *Pullularia pullulans* and two *Polyspora lini*) another. The similarity of all the other characters, however, and the serological compatibility of all six cultures would suggest that they are generically identical. Thus, *P. lini* is considered really to be a *Pullularia*, and the sole specific difference found in this work is the sprout-cell shape and colony colour on potato slope. It is suggested that it might be referred to as *P. pullulans* var. *lini*.

If *Polyspora* and *Pullularia* are identical, their association with flax browning must be secondary. The suggestion is made that browning symptoms may develop as a result of attack by the *Pullularia*, which develops on the flax together with associated fungi when the plant is adversely affected by environmental conditions. The author's failure to obtain any pathogenicity reaction on flax suggest that a very special set of environmental conditions may be necessary to induce browning.

It is pointed out that, in spite of seed treatments, browning has been reported as occurring in areas that never produced flax before: this supports the probable

identity of *Polyspora* and *Pullularia*, and the latter being ubiquitous, seed treatment would be useless. In the present work, *Pullularia* was found to occur at about the same rate on seed from both healthy and diseased crops. The beneficial effect of seed treatment on germination in Petri dish cultures would appear to result from the removal of saprophytes. *Pullularia* was also found to be inimical to the germination of grass and peas, and mycelium of the fungus in the seed coats of grass and peas was ascertained to be non-pathogenic.

Angell (H. R.). **Browning of Flax and excess soil moisture.**—*J. Coun. sci. industr. Res. Aust.*, xviii, 2, pp. 150–152, 1945.

In March, 1943, the author investigated the prevalence of a flax disease apparently indistinguishable from browning [see preceding abstract] in Tasmania. As the condition was general over 250 acres of peaty soil in five different fields and was not observed in any other fields sown with seed from the same source, it appeared to be, probably, due to some adverse soil condition. In December of the same year it was present in two fields near Calac, Victoria. It had also been observed at Canberra, A.C.T., in 1942. Plantings from the roots of the Canberra plants gave only saprophytes.

In an experiment made outdoors at Canberra, with over 200 4-gal. drums sown with Liral Crown on 14th to 15th August, 1944, and subjected to various treatments, symptoms of browning developed on 15th November on the upper part of one plant in a waterlogged drum. The water on the surface of the soil in this and other waterlogged drums was at once drained off, and from that time until harvesting a normal amount was supplied. The plants in every drum in which the soil had been waterlogged in turn developed browning. The most conspicuous feature was the general browning and dying of individual plants during the flowering and fruiting season. Only in one other drum did a few plants show browning; in this, the soil-level was well below the brim; and this had, apparently, led to overwatering. There was little difference between the fibre obtained from the controls and the waterlogged plants.

After the results of this experiment were known, the author observed that browning also began in the field after the excess soil water had disappeared. It should not, however, be assumed from this work that the disease is always associated with excess soil water. The physical and chemical composition of the soil appear to exercise a controlling influence.

So far, browning has occurred in only a few fields in Australia, and whether or not it is of economic importance is doubtful. The available evidence suggests that if a crop is otherwise satisfactory it should not be degraded because of browning.

Machacek (J. E.) & Brown (A. M.). **Threshing-injury to Flax seed in Canada.**—*Sci. Agric.*, xxv, 10, pp. 601–625, 4 figs., 1925.

The results of more detailed, but often somewhat inconclusive, experiments on the prevalence and effects of threshing injury to flax seed in Canada, suggest to the authors of this paper that the crop losses resulting from it are less severe than indicated by earlier investigations [*R.A.M.*, xxii, p. 98]. Dry weather makes seed brittle and, while it never cracks when hand-threshed or passed through rubber rollers, more or less severe fracturing occurs when it is treated by machines equipped with a steel threshing cylinder. Flax seed of relatively high water content is in consequence less brittle and, being also protected to some extent by the spongy material from moist bolls, is less susceptible to injury. This is probably why threshing injury seems less prevalent in eastern than in western Canada, where the average of cracked seed during the years 1940 to 1943 was over 40 per cent. Large-seeded varieties seem more liable to damage than those with small seeds.

Fractured kernels, planted in ordinary soil, are subject to attack by soil-borne micro-organisms. These attacks are usually more serious in heavy soils than in light, but variations in soil temperature do not appear to affect the frequency of infection. It was noticed that rotting of fractured kernels under field conditions was less severe than in greenhouse experiments. Though seed treatment, notably with 1½ oz. ceresan per bush. on heavy soils, improved the germination of fractured seed, improved yields did not always result, and while uninjured field-sown seed germinated better than fractured kernels, there was often nothing to choose between the yields. Overdoses of disinfectants retarded germination and the seedlings often showed enlarged cotyledonary leaves and sometimes no others. Damaged seed was not only liable to further injury from disinfectants, but the ill effects of this were observed also in the seedlings. An effect of sowing seed, whose germinating capacity had been improved by disinfection, at the normal rate of seeding practised, was that more plants than could be adequately nourished were produced and the yield consequently was not likely to prove any greater than from poorer and untreated seed sown at the same rate.

Da Silva Fernandes (C.). **O rolamento do broto do Sisal (Agave sisalana Perrine).** [Sisal (*Agave sisalana* Perrine) bud roll.] -*Bol. S.A.I.C.*, xi, 1–2, pp. 60–63, 10 figs., 1944.

Sisal in the State of Parahyba, north-eastern Brazil, suffers from a deficiency of potassium, expressed as leaf basal necrosis as described by Medina [*R.A.M.*, xxiii, p. 487], and from calcium deficiency [rolamento] manifested by a complete severance of the leaves near their base, almost invariably at right angles to the cone formed by the leaves of the bud. The surfaces exposed by this process present a blackened appearance corresponding to an intense suberization of the severed tissues. The proximal ends remain healthy and continue to develop to a normal size, while from the centre of the circle formed by the transverse truncation of the bud arise perfect new leaves. The disturbance, however, appreciably retards the growth of the plant, and the procurement of fibre from the broken distal portions is impracticable or uneconomic. The symptoms appear to reach a climax some time after the first sharp showers following a hot, protracted summer. There is no reason to believe that the disorder, which also affects henequen (*Agave fourcroides*), is of parasitic origin.

Frick (Louise). **Untersuchungen über die Biologie und Pathogenität von Diplocarpon rosae (Lib.) Wolf.** [Studies on the biology and pathogenicity of *Diplocarpon rosae* (Lib.) Wolf.]—*Phytopath. Z.*, xiv, 6, pp. 525–591, 2 pl., 13 figs., 1 diag., 5 graphs, 1943. [Received September, 1945.]

Evidence is adduced for the recognition of the rose black-spot fungus, *Diplocarpon rosae* [*R.A.M.*, xi, p. 792; xiii, p. 771; *et passim*] in Switzerland as early as 1894. Hitherto little attention has been paid to its occurrence on wild roses, among which the author found the following species affected: *Rosa arvensis*, *R. canina*, *R. glauca*, *R. pomifera*, *R. eglanteria*, *R. elliptica*, *R. dumetorum*, *R. hemitricha*, *R. thuilleri*, *R. uriensis* [*R. glauca*], *R. vogesiaca*, and *R. foetida*.

The organism is a facultative parasite, the hyphae of which spread through the subcuticular tissues of the host, as well as intercellularly in the interior of the living leaves and intracellularly in the saprophytic phase. The black colour of the foliar lesions is attributable to the dark shade of the subcuticular hyphae and of the disorganized host cells. The bicellular, hyaline, oblong to clavate conidia produced during the summer in the subcuticular acervuli measure (average of three isolates) 15·5 to 16·9 by 4·6 to 5·4 μ, the corresponding dimensions for those developing during the winter in the subepidermal pycnidia in dead leaves being 17·2 to 19·2 by 4·8 to 5·8 μ. The subcuticular acervuli of the summer phase

further give rise during the autumn to unicellular, hyaline, bacteroid microconidia, 2·5 to 4·5 by 1 to 2 μ, which are abstricted from bicellular conidiophores superficially resembling macroconidia, but narrower, shorter, and not constricted at the septum.

Summer and winter conidia from rose leaves and malt agar cultures germinated in the same manner, in hanging drops, each conidium producing up to five germ-tubes. The minimum, optimum, and maximum temperatures for the process were found to be 0°, 18°, and 33° C. Only 2 per cent. of the conidia collected on diseased leaves on 6th March, 1942, germinated compared with 97 per cent. of those dating from 2nd May, the corresponding figures for 1st June, 6th July, 17th August, and 12th and 30th October being 87, 26, 31, 43, and 70, respectively. Three- and six-day-old conidia germinated to the extent of 92 and 88 per cent., respectively, while from 9 to 27 days the figure dropped from 71 to 6, and by the 31st day viability was altogether lost. A synthetic auxin, 'Roche 202', was shown to stimulate germination. On leaves of the W. E. Chaplin variety, the germination process differed in certain features from that in hanging drops, beginning later (after 18 compared with 9 hours) and only one short germ-tube being normally produced. The single germ-tube penetrates the intact cuticle or occasionally (on the under side) enters the leaf through the stomata.

In Switzerland, and apparently in other European countries, the fungus occurs exclusively in the conidial phase, *Marssonina rosae*, also known as *Actinonema rosae* [ibid., iv, p. 672], the connexion between which and *D. rosae* was demonstrated by Wolf (*Bot. Gaz.*, liv, pp. 218-234, 1912). Overwintering is effected by means of the saprophytic mycelium, and re-infection in the spring takes place mostly from the dead leaves strewn over the soil, the conidia produced at this season in the subepidermal pycnidia being splashed by rain on to the young basal leaves.

Under uniform environmental conditions *D. rosae* infects both leaf surfaces with equal severity, but further growth is better on the upper than on the lower side. Young leaves are more rapidly and virulently attacked than old ones, and one- and two-year-old seedlings are more susceptible than plants of several years old. Very young leaves are protected from infection by a coating of wax. In inoculation experiments with 12 isolates of the fungus on two cultivated varieties, W. E. Chaplin and Etoile de Hollande, and seven wild species, one isolate proved to be non-pathogenic: otherwise there were no indications of physiologic specialization. Varying reactions to infection, however, were shown by the wild roses, *R. cinnamomea* and *R. pendulina* being entirely and *R. arvensis* virtually immune while *R. foetida* was the most susceptible, followed by *R. eglanteria*, *R. glauca*, and *R. pomifera* in the order given. Negative results were obtained in inoculation tests on Rosaceae other than the rose.

The fungus rapidly loses its pathogenicity in pure culture; after seven months three isolates from wild roses caused no infection on Etoile de Hollande and only a low percentage (maximum of 7·9) on W. E. Chaplin after five weeks' incubation. The same two varieties contracted 33·7 to 44·3 and 43·6 to 49·8 per cent. infection, respectively, when inoculated with fresh summer conidia. *D. rosae* grows very slowly, 15 to 37 days elapsing before the incipient mycelium could be discerned, and the diameter of the colonies after a month ranging from 2 to 9 mm. However, among 18 isolates studied (13 from cultivated and 5 from wild roses), the differences in the mycelial growth rate were apparent, the diameters varying between 3·9 (Betty Uprichard) and 15·7 mm. (Mrs. Sam McGredy). The temperature relations of the various isolates were identical, the minimum, optimum, and maximum for development being 0°, 21°, and 30°. The slight disparities in the individual means of the conidial dimensions of isolates from 16 cultivated and 10 wild roses failed to provide a basis for separation into morphological races.

The problems connected with control by breeding for resistance [*R.A.M.*, xix, p. 540] and chemical treatments [ibid., xxiv, p. 103] are briefly discussed.

Cochrane (V. W.). **The effect of artificial light on germination of urediospores of Phragmidium mucronatum (Fr.) Schlecht.**—*Phytopathology*, xxxv, 6, pp. 458–462, 1945.

Under artificial light from 1,250-ft. candles, both the germination rate and final germination of rose rust (*Phragmidium mucronatum*) uredospores [*R.A.M.*, xxiv, p. 417] were depressed, the germination percentages after three, six, and 24 hours at 19·5° to 20° C. being 7, 28·6, and 42·2, respectively, as against 85, 92·9, and 93·7, respectively, for the unilluminated controls. A test of germination in darkness of previously illuminated spores showed that only 21 per cent. were viable after 24 hours' exposure to light. The apparently lethal effects of strong light may, however, be only indirect. Spores prevented from germination by light, but otherwise uninjured, may have lost their viability through the leaching of soluble cell constituents into the agar medium. Unpublished data showed that spores on agar rapidly lose their viability if prevented from germinating by low temperatures; in one test, 24 hours' storage of spores on agar at 3° resulted in a decline of viability from 91·1 to 25·4 per cent. The subsequent germinability of dry spores was not impaired by 24 hours' exposure to light of an intensity of 1,250-ft. candles. In this connexion it may be noted that the intensity of natural light at mid-day in summer often reaches the value of 10,000–12,000-ft. candles. Illumination from 200-ft. candles caused only slight temporary delay of germination, while light of low intensity (2·4-ft. candles) exerted no effect on the process.

Smith (K. M.). **Dahlias and the problem of virus infection.**—*Gdnrs' Chron.*, Ser. 3, cxviii, 3066, pp. 140–141, 2 figs., 1945.

Notes are given on the symptoms and control of three viruses affecting the *Dahlia* in England, viz., tomato spotted wilt, cucumber mosaic, and *Dahlia* mosaic. The first-named produces well-defined rings on some varieties, such as Brentwood Bedder, but on others the symptoms are somewhat indeterminate, though a ring-like tendency is apparent. The cucumber mosaic virus has only recently been isolated from *Dahlia* plants at the Plant Virus Research Station, Cambridge. The leaves bear mottled, pale and dark green patches, without blistering, accompanied in some varieties by green whorls or ring-and-line patterns distinct from the clear-cut, concentric rings typical of spotted wilt. Yet other varieties, such as Bishop of Llandaff, serve as symptomless carriers of the virus. Unlike the two foregoing viruses, with their numerous hosts, the *Dahlia* mosaic virus is apparently confined to the one genus. Rings do not develop on the leaves, which show a yellowish-green banding of the veins. Some varieties show foliar distortion and blistering and there is a tendency for the lateral shoots to be shortened, producing bushy, stunted plants. Control of the viruses should be based on the suppression of aphids and thrips by spraying, dusting, or fumigation with nicotine, and the building-up of healthy nucleus stocks, which should be isolated in a special glasshouse and regularly rogued.

Hollowell (E. A.). **Registration of varieties and strains of Alfalfa, I.**—*J. Amer. Soc. Agron.*, xxxii, 8, pp. 649–652, 1945.

Ranger (*M*[*edicago*] *media*), a synthetic lucerne variety (Reg. No. 1) originated and developed at the Colorado and Nebraska Agricultural Experiment Stations, respectively, is resistant to bacterial wilt (*Corynebacterium insidiosum*) and winter-hardy. At the Iowa Agricultural Experiment Station in 1943, when stand reductions were caused by the disease, Ranger yielded 3·55 tons of hay per acre,

compared with 2·47, 3·47, 1·75, and 3·01 for Grimm, Hardistan, Kansas Common, and Ladak, respectively.

Buffalo (Reg. No. 2) is a highly wilt-resistant, purple-flowered selection from Kansas Common developed at the Kansas Agricultural Experiment Station.

Meeker Baltic (Reg. No. 3), a natural selection from Baltic, is susceptible to wilt and therefore adapted for cultivation in the northern and central States where the disease is not a limiting factor.

Black (L. M.). **A virus tumor disease of plants.** —*Amer. J. Bot.*, xxxii, 7, pp. 408–415, 9 figs., 1945.

Pursuing his investigations into the host range and symptomatology of the new plant virus, *Aureogenus magnivena* [*R.A.M.*, xxiii, p. 490], for which the name 'wound-tumour virus' (causing 'wound-tumour disease') is now suggested as an alternative to 'clover big-vein virus', the author describes experiments which show the pathogenicity of the virus, isolated from *Agallia constricta* leaf-hoppers, to 43 suscepts from ornamental, vegetable, and common weed plants, the following species proving notably easy to infect: *Brachycome iberidifolia*, *Chrysanthemum leucanthemum* var. *pinnatifidum*, *Melilotus alba*, *Trifolium incarnatum*, *Rumex acetosa*, *Linaria maroccana*, and *Schizanthus wisetonensis*.

In the wound-tumour disease the presence of the virus is manifested not by the more usual symptom of the 'clearing of the veins', but by an enlargement of the veins on the lower surface of the leaf, frequently accompanied by the depression of the vein below the upper surface of the lamina, and giving rise to either enations, as exemplified by observations on *Vinca rosea*, *T. incarnatum*, and *Lobelia erinus*, where they are large with nodular basal clusters, or to vein-tumours which protrude on the under side of leaves through the green surface tissues to expose the white tumour tissue, as exhibited in the cases of *R. acetosa* and *C. leucanthemum*. Diseased leaves commonly show various kinds of distortion, dwarfing, and curling, the tops of the plants being severely stunted in many instances. *M. alba* alone showed tumours both on stems and roots, while the leaves showed no symptoms other than slight dwarfing. The response of individual representatives of a species to infection, as in the case of *R. acetosa*, often varies considerably.

The tumours produced by the virus on the roots of many suscepts are characterized by overgrowths tending to become spherical and woody. According to the species of suscepts, the size ranges up to 1 cm. in diameter and on some roots were so numerous that they became fused together. Other species showed only scattered tumours, while in yet others a cluster of tumours appeared on a single lateral root. In one species the root tumours were a deep purple, although the normal root was almost white. It seemed that a definite connexion existed between the root tumours and the emergence points of the lateral roots. Tumours were also produced on the crown of certain hosts and on the petioles of others. Wound-tumour disease resembles Fiji disease of sugar-cane [ibid., xiii, p. 539]; possibly wallaby ear-disease of maize [ibid., xxi, p. 484]; in some respects crown gall (*Bacterium tumefaciens*); and may be related to curly top of beets in Argentina. The author believes that further study of the pathogen will throw more light on the virus causation of plant tumours.

Fischer (G. W.). **The mode of infection and the incubation period in the stem smut of grasses, Ustilago spegazzinii (U. hypodytes).**—*Phytopathology*, xxxv, 7, pp. 525–532, 1945.

In inoculations with spore suspensions of *Ustilago spegazzinii* and its var. *agrestis* (*U. hypodytes*) [*R.A.M.*, xxiv, p. 420] from several species of *Agropyron* and *Elymus*

at the Washington Agricultural Experiment Station, positive results were obtained only on mature plants of *A. cristatum*, *A. trachycaulum*, and *E. canadensis*, seed and blossom tests being unsuccessful. Infection probably takes place through the meristematic tissues but the minimum incubation period was two years, a trace of stem smut developing in the second heading after inoculation and becoming abundant in the third. *A. repens*, one of the hosts from which the inoculum for the above-mentioned experiments was procured, is believed to constitute the source of the common local outbreaks of the disease. Once established in a stand of a susceptible species, *U. spegazzinii* tends to increase rapidly year after year, denoting that spread occurs from plant to plant. The most practicable method of control will probably consist in the use of resistant strains of forage grasses, but owing to the lengthy incubation period of smut it is unlikely to appear until three years after planting or, under normal conditions, to limit production to any extent for two or three years more, i.e., five or six from the time of planting.

Sprague (R.) & Johnson (A. G.). **Selenophoma on grasses. II.**—*Mycologia*, xxxvii, 5, pp. 638–639, 1945.

The genus *Selenophoma* is emended by the authors to include species with somewhat obtusely pointed spores, other characters being concordant. *S. obtusa* is described as a new species with conidiophores measuring 40 to 150 × 40 to 138 μ and conidia 13 to 17 × 2·5 to 4·2 μ. It is found on living leaves and culms of various *Sitanion*, *Stipa*, *Poa*, *Elymus*, and *Agropyron* grasses in the western United States. *Phyllosticta stomaticola* is renamed *Selenophoma donacis* var. *stomaticola* (Baüml.) n. comb. and *Septoria everhartii* becomes *Selenophoma everhartii* (Sacc. & Syd.) n. comb.

Olive (L. S.). **A new Dacryomyces-like parasite of Arundinaria.**—*Mycologia*, xxxvii, 5, pp. 543–552, 35 figs., 1945.

Specimens of a leaf parasite of *Arundinaria tecta*, representing a new genus and species to which it is proposed to give the name *Dicellomyces gloeosporus*, were received at the United States Department of Agriculture from Georgia and North Carolina. The fungus causes small, yellow spots which enlarge and become dark brown, usually with a surrounding yellow zone. Small, yellow fructifications appear on the lower surface of the spots and eventually large portions of the leaves may become brownish and dead. The young sporocarp originates within the leaf and ruptures the epidermis. The fungus resembles *Dacryomyces* in colour, form of fructification, gelatinous nature, 2-sterigmate basidia, and allantoid basidiospores (8·6 to 11·9 by 2·7 to 4·5 μ), which may become septate and frequently bud out globose conidia. The persistent probasidia, basidia produced externally to the gelatinous matrix, and relatively short sterigmata distinguish this parasite from all other genera of the Dacryomycetaceae.

Union of South Africa Proclamations Nos. 116 and 117, 1945. Restriction on the introduction of certain plants into the Union. Portions and products of certain plants included in the definition of plant.—2 pp., Govt. Printer, Pretoria, 1945.

These Proclamations, dated 28th and 25th May, 1945, respectively, introduce amendments into the Schedule to Proclamation No. 286 of 1936 [*R.A.M.*, xvi, p. 640] involving the prohibition of importation into the Union of South Africa of seeds, plants, logs, or unmanufactured timber of any species of elm or *Zelkova* from countries harbouring the Dutch elm disease (*Ceratostomella ulmi*). The restriction likewise applies to articles made from these woods, e.g., packing-cases or barrels, on which bark is present.

COMMON NAMES OF VIRUS DISEASES USED IN THE REVIEW OF APPLIED MYCOLOGY

The attitude of the *Review of Applied Mycology* to questions of nomenclature is conservative. In the interests of continuity changes in the scientific names of pathogens are made as infrequently as possible. The fungus names included in the British Mycological Society's *List of Common British Plant Diseases* are accepted, while the names adopted for other fungi are those recognized by responsible pathologists specializing in the particular diseases concerned. Recently a notable clarification of the confusion in bacterial nomenclature was effected, after consultation with bacteriologists at home and abroad, by adopting the genera *Pseudomonas*, *Xanthomonas*, and *Corynebacterium*, but the nomenclature of viruses remains a problem of great complexity. The *Review* has consistently refrained from adopting any system for the 'scientific' nomenclature of viruses since no system yet invented has obtained international acceptance. In the past a virus was indexed in the *Review* either under the English common name of the *disease* it causes or under some other widely used designation.

The common names for many diseases are accepted throughout the English-speaking world, they are frequently without ambiguity, and they will presumably continue to be used after the viruses have received internationally acceptable scientific names. It was decided, therefore, that, with one or two exceptions, viruses should be indexed in the *Review* under common names derived from the English common names of the diseases they cause. To this end an effort is being made to standardize disease names.

In March, 1943, a preliminary list of about 120 disease names was circulated for comment to a number of virus workers and official plant pathologists, whose suggestions were incorporated in an expanded list which was published in mimeographed form on 1st January, 1944. The names in the latter list have since been scrutinized by interested workers in all parts of the English-speaking world, and there appears to be very general agreement on the use of the disease names given in the present list. In certain cases where there are two well-established common names for one disease (e.g., maize mosaic and corn mosaic) the one selected for making the common name of the virus for use in the *Review* is followed by the other as a synonym. The diseases included in the present list have all been definitely proved to be of virus origin and all merit standard common names. There are, however, many other virus, or possibly virus, diseases recorded in the literature, but until more is known about them it is felt to be best not to recommend common names.

Under each disease name appears the name of the causal virus used for indexing purposes in the *Review*. In the 1944 mimeographed list an attempt was made to give the full synonymy for each virus, for there was then no publication in which complete lists of synonyms were available. During the past eighteen months, however, the various Sub-Committees of the Committee on Virus Classification and Nomenclature of the American Phytopathological Society have been publishing as a series of supplements to the *Plant Disease Reporter* summaries of viruses described on different plants or groups of plants and giving the full synonymy for each virus dealt with. Many of the earlier synonyms for most of the important viruses are of interest only to the virus specialist, who is referred to the series of publications mentioned, but for the convenience of the general plant pathologist the important synonyms for each virus have been given in this list where an effort

has been made to distinguish the 'obligate' from the 'facultative' synonyms (see Ainsworth, *Ann. appl. Biol.*, xxx, 183, 1943).

For each disease reference is made to a description in a standard textbook or other work. The full citation for a name attributed to an author and date can be obtained by consulting the works listed below, the bibliographies of Atanasoff (1934, supplements *Phytopath. Z.*, x, pp. 339–463, 1937; ibid., xii, pp. 511–84, 1940) and M. T. Cooke (*J. Agric. Puerto Rico*, 1934–8), or the *Review of Applied Mycology*.

The insertion of 'virus' or an author's name in square brackets indicates that the author used the name but did not recognize the virus nature of the disease. A virus name derived from a disease name is attributed to the author using the disease name and recognizing that a virus was involved whether or not he actually used the common name of the virus.

The mimeographed list published in 1944 was submitted to the Special Committee on Virus Nomenclature and Classification of the American Phytopathological Society and to the Plant Pests and Diseases Committee of the Association of Applied Biologists for their comments, and I am grateful to these bodies for so carefully examining the American and British names and making many valuable suggestions. I am also indebted for suggestions, encouragements, and advice to Professor R. E. D. Baker (Trinidad), Mr. F. C. Bawden (Rothamsted), Dr. C. O. Bennett (Chairman of the Special Committee of the American Phytopathological Society), Dr. L. M. Black (Rockefeller Institute), Dr. L. C. Cochran (United States), Dr. G. H. Cunningham (New Zealand), Professor H. S. Fawcett (California), Dr. C. E. Foister (Scotland), Dr. R. V. Harris (East Malling), Dr. E. M. Hildebrand (United States), Dr. F. O. Holmes (Rockefeller Institute), Dr. J. C. Hopkins (Southern Rhodesia), Professor J. Johnson (Secretary of the Committee on Description and Nomenclature of Plant Viruses of the International Botanical Congress), Dr. W. G. Keyworth (Secretary of the Plant Pests and Diseases Committee), Dr. R. McKay (Eire), Professor A. E. Muskett (Northern Ireland), Dr. G. Watts Padwick (India), Mr. I. W. Prentice (East Malling), Mr. G. Samuel (England & Wales), Dr. I. W. Selman (Cheshunt), Dr. K. M. Smith (Cambridge), Dr. H. H. Storey (Amani Institute), and Dr. F. Weiss (United States). Finally, I should like to make special mention of the considerable editorial assistance given by Dr. G. C. Ainsworth in the preparation of this list.

S. P. Wiltshire
Editor

21*st December*, 1945

ABBREVIATED CITATIONS USED IN THIS LIST

Smith *Tb.*	Smith (K. M.), *Textbook of plant virus diseases*, 1937.
Holmes *Hb.*	Holmes (F. O.), *Handbook of phytopathogenic viruses*, 1939.
Hildebrand *et al.*	Hildebrand (E. M.), Berkeley (G. H.), and Cation (D.), *Handbook of virus diseases of stone fruits in North America*, 1942 (Mich. Agric. Exp. Sta.).
Brierley *P.D.R.S. 150*	Brierley (P.), Virus diseases described primarily on ornamental or miscellaneous plants, *Plant Disease Reporter, Suppl.* 150, 1944.
Weiss *P.D.R.S. 154*	Weiss (F.), Viruses described primarily on leguminous vegetable and forage crops, *ibid.*, 154, 1945.
Weiss *P.D.R.S. 155*	Weiss (F.), Viruses, virus diseases, and similar maladies of potatoes, *Solanum tuberosum* L., *ibid.*, 155, 1945.
Brierley *P.D.R.S. 158*	Brierley (P.), Viruses described primarily on ornamental and miscellaneous plants, II, *ibid.*, 158, 1945.

Abaca Bunchy Top. Smith *Tb.* 408; Holmes *Hb.* 63; Brierley *P.D.R.S. 158*, 185.

Abaca bunchy top virus. Ocfemia 1932.
Manila bunchy top virus. Ocfemia 1932.
Musa virus 2. Smith *Tb.*
Marmor abaca Holmes *Hb.*

Abutilon Variegation. Smith *Tb.* 599; Holmes *Hb.* 50; Brierley *P.D.R.S. 150*, 412.

Abutilon variegation virus. [Morren 1869] Brierley 1944.
Abutilon infectious variegation virus [virus]. Bauer 1904.
Abutilon infectious chlorosis [virus]. Bauer 1906.
Abutilon virus 1. Smith *Tb.*
Marmor abutilon Holmes *Hb.*
Abutilon mosaic virus. Holmes *Hb.*

Agropyron Mosaic. McKinney, *J. Wash. Acad. Sci.*, xxxiv, 326, 1944.

Agropyron mosaic virus.
Marmor agropyri McKinney 1944.

Strains or suspected strains.

Agropyron green-mosaic virus. McKinney 1944.
Marmor agropyri var. *typicum* McKinney 1944.
Agropyron yellow-mosaic virus. McKinney 1944.
Marmor agropyri var. *flavum* McKinney 1944.

Alfalfa, see Lucerne.

Anemone Alloiophylly. Smith *Tb.* 6; Holmes *Hb.* 108; Brierley *P.D.R.S. 150*, 415.

Anemone alloiophylly virus. Klebahn 1926 [as 'alloiophyllie'].
Anemone virus 1. Smith *Tb.*
Galla anemones Holmes *Hb.*

Apple Mosaic. Smith *Tb.* 150; Holmes *Hb.* 75.

Apple mosaic virus. Holmes *Hb.*
Apple infectious variegation virus. Bradford & Joly 1933.
Pyrus virus 2. Smith *Tb.*
Marmor mali Holmes *Hb.*

Aster Yellows. Smith *Tb.* 214; Holmes *Hb.* 2 (host list, p. 170); Brierley *P.D.R.S. 150*, 419; *ibid. 158*, 169.

Aster yellows virus. Kunkel 1924, 1936.
Lettuce Rio Grande disease [virus]. R. E. Smith 1902 fide Kunkel 1925.
Lettuce rabbit-ear virus. fide Kunkel 1925.

Lettuce white heart virus. fide Kunkel 1926.
Salsify yellows virus. Haskell & Archer 1929.
Erigeron yellows virus. McClintock 1931.
New York aster yellows virus. Kunkel 1932.
Gelbsucht der Sommerastern virus. Richter 1936.
Callistephus virus 1. Smith *Tb.*
Chlorogenus callistephi var. *vulgaris* Holmes *Hb.*
Aster yellows virus, typical strain. Holmes *Hb.*
Lettuce and endive yellows virus. Linn 1940.
Potato purple-top-wilt virus. [Brentzel 1938] fide Younkin 1943

Strains or suspected strains
Aster yellows virus, heat attenuated strain. Holmes *Hb.*
Chlorogenus callistephi var. *attenuatus* Holmes *Hb.*
California aster yellows virus. Kunkel 1932. Severin 1936.
Celery yellows virus. Severin 1929.
Carrot, parsnip and parsley yellows virus. Severin 1930, 1932.
Callistephus virus 1A. Smith *Tb.*
Chlorogenus callistephi var. *californicus* Holmes *Hb.*
Aster yellows virus, celery yellows strain. Holmes *Hb.*
Delphinium aster yellows virus. Severin & Oliver 1936.

Banana Bunchy Top. Smith *Tb.* 404; Brierley *P.D.R.S.* 118, 187.
Banana bunchy top virus. Magee 1927.
Plantain bunchy top virus. [Bryce 1927] fide Hanson & Park 1936.
Banana virus 1. J. Johnson in Smith *Tb.*
Musa virus 1. Smith *Tb.*

Bean Leaf Wilt. J. Johnson, *J. agric. Res.*, lxiv. 448, 1942.
Bean leaf wilt virus. J. Johnson 1942.

Bean Mosaic. Smith *Tb.* 136; Holmes *Hb.* 87; Weiss *P.D.R.S.* 154, 43 (gives a longer list of synonyms).
Bean mosaic virus. Stewart & Reddick [1917] 1919.
Common bean mosaic virus. Pierce 1934.
Typical bean mosaic virus. Nelson 1932.
Bean virus 1. Pierce 1934.
Phaseolus virus 1. Smith *Tb.*
Marmor phaseoli Holmes *Hb.*
Ordinary bean mosaic virus. Costa & Forster 1941.
Strain or suspected strain.
Bean mosaic virus, "new strain." Sachanis & Burkholder 1943

Bean Mosaic, Southern. Zaumeyer & Harter *J. agric. Res.*, lxvii. 305–325, 1943; Weiss *P.D.R.S.* 154, 46.
Southern bean mosaic virus. Zaumeyer & Harter 1943.
Southern bean mosaic virus 1. Zaumeyer & Harter 1943.
Bean mosaic virus 4. Zaumeyer & Harter 1942.
Marmor laesiofaciens Zaumeyer & Harter 1943.
Strain or suspected strain.
Southern bean mosaic virus 2. Zaumeyer & Harter 1943.
Bean mosaic virus 4A. Zaumeyer & Harter 1943.
Marmor laesiofaciens var. *minor* Zaumeyer & Harter 1943.

[illegible] Yellow Mosaic. Smith *Ti.* 31 | Wade *P. D. S. A.* 134, 67.

Bean yellow mosaic virus. Pierce 1934.

Bean virus 2. Pierce 1934.

[illegible] Zaumeyer & Wade [1931-1937]. Ide Wade 1937.

[illegible] Zaumeyer & Wade 1937 Ide Wade 1942.

Phaseolus virus 2. Smith *Ti.*

Bean dwarf mosaic virus. Costa & Forster 1941.

[illegible]

Pea mosaic virus 2. Zaumeyer & Wade 1935.

Pea mosaic virus 4. Zaumeyer 1940.

Pisum virus 4A. Wade 1936.

Pea crust mosaic virus. Zaumeyer 1940.

Pea mosaic virus 5. Zaumeyer 1940.

Pisum virus 6. Wade 1936.

Pea virus 2. Orton 1937.

Pisum virus 4. Wade 1936

[illegible]

[illegible] Johnson, *J. agric. Res.* [illegible] 1942

Bean yellow necrosis virus. J. Johnson 1942.

[illegible] Leaf Curl. Smith *Ti.* 42; Holmes *Hb.* 132.

Sugar beet leaf curl (Amer.).

Beet leaf curl virus. [Wille 1928.]

Sugar beet leaf crinkle [virus]. Wille 1928.

Rübenkopf [virus]. Wille 1928 fide Holmes *Hb.*

Sugar beet virus 3. J. Johnson in Smith *Ti.*

Beta virus 3. Smith *Ti.*

Betula latus Holmes *Hb.*

Beet Kräuselkrankheit virus. Holmes *Hb.*

Beet Curly Top. Smith *Ti.* 21; Holmes *Hb.* 11 Cont list p. 174).

Sugar beet curly top (Amer.).

Beet curly top virus. [Hull 1929; Bennett & Hoover 1931] Carsner [Ark. Invest. 1933]

Tomato yellows [virus]. Humphrey 1925.

Western yellow blight virus.

Beta virus 1. Smith *Ti.*

Sugar beet virus 1. J. Johnson in Smith *Ti.*

Chlorogenus borealis Holmes *Hb.*

Rugia verrucosans Carsner & Bennett 1943.

[illegible] or suspected strains.

Attenuated sugar beet curly top virus. Carsner 1925, Bennett 1935.

Sugar beet curly top (Argentina) virus. Fawcett 1927.

Sugar beet curly top virus strains No. 1, 2, 3, 4. Giddings 1937.

Beet Mosaic. Smith *Ti.* 37; Holmes *Hb.* 72.

Sugar beet mosaic (Amer.).

Beet mosaic virus. [Rabbow 1927; Wiegan 1928.]

Beet yellows [virus]. Prillieux & Delacroix 1896 fide Smith *Ti.*

Beet mosaic virus. Lind 1915.

[illegible] and beet mosaic virus. Pierce 1927.

Beet [illegible] virus. Potter 1930 fide Holmes *Hb.*

Sugar beet virus 2. J. Johnson in Smith *Ti.*

Beta virus 2. Smith *Tb.*
Marmor betae Holmes *Hb.*

Beet Savoy Disease. Smith *Tb.* 47; Holmes *Hb.* 132.
Sugar beet Savoy disease (Amer.).
Beet savoy disease virus. Coons, Kotila, & Stewart 1937.
Beta virus 5. Smith *Tb.*
Savoia piesmae Holmes *Hb.*
Beet savoy virus. Holmes *Hb.*

Beet Yellows. Smith *Tb.* 45; Watson, *Ann. appl. Biol.*, xxix, 358–365, 1942.
Sugar beet yellows (Amer.).
Beet yellows virus. Watson 1942; Petherbridge & Stirrup 1935.
Beet vergelingsziekte virus. Quanjer 1934.
? Beet jaunisse virus. Roland 1936 fide Smith *Tb.*
Beta virus 4. Smith *Tb.*

Blackberry Dwarf. Smith *Tb.* 124; Holmes *Hb.* 124.
Blackberry dwarf virus. Zeller 1927.
Loganberry dwarf virus. Zeller 1925, 1927.
Dewberry dwarf virus. Zeller 1927.
Raspberry virus 2D. J. Johnson in Smith *Tb.*
Rubus virus 5. Smith *Tb.*
Nanus loganobacci Holmes *Hb.*

Brome Mosaic. McKinney, *J. Wash. Acad. Sci.*, xxxiv, 325–326, 1944; *Phytopathology*, xxiv, 993, 1944.
Brome mosaic virus. McKinney 1944.
Bromus inermis mosaic virus. McKinney, Fellows, & Johnston 1942.
Marmor graminis McKinney 1944.

Cabbage Ring Necrosis. Larson & Walker, *J. agric. Res.*, lxii, 475–491, 1941.
Cabbage ring necrosis virus. Larson & Walker 1941.

Cabbage Black Ring Spot. Smith *Tb.* 6 [? caused by a strain of Turnip mosaic virus (q.v.)].
Cabbage black ring spot virus. Smith 1935.
Wallflower mosaic virus. Smith 1935.
? Cabbage blackspot virus. Tompkins 1935 fide Smith *Tb.*
Brassica virus 1. Smith *Tb.*
Cabbage black ring virus. Tompkins 1938 fide Smith *in litt.*
Cabbage mosaic virus. Larson & Walker 1939 (in part) fide Smith *in litt.*
Turnip mosaic virus. Chamberlain 1936 fide Prentice *in litt.*

Cacao Swollen Shoot. Posnette, *Trop. Agriculture, Trin.*, xviii, 87–90, 1941; Brierley *P.D.R.S. 158*, 198.
Cacao swollen shoot virus. [(W. F. Stevens) 1937] Posnette 1940.

Cassava Brown Streak. Brierley *P.D.R.S. 158*, 180.
Cassava brown streak virus. Storey 1936.
Cassava stem lesion virus. Storey 1936.

Cassava Mosaic. Smith *Tb.* 94; Holmes *Hb.* 117; Brierley *P.D.R.S. 158*, 181.
Cassava mosaic virus. Lefevre 1935, Storey & Nichols 1938.
Cassava 'Kräuselkrankheit' [virus]. Warburg 1894.
Manihot virus 1. Smith *Tb.*

Ruga bemisiae Holmes *Hb.*
Ochrosticta bemisiae (Holmes) McKinney 1944.

Cauliflower Mosaic. Smith *Tb.* 13; Holmes *Hb.* 69.
Cauliflower mosaic virus. Tompkins 1937.
Brassica virus 3. Smith *Tb.*
Marmor cruciferarum Holmes *Hb.*
Cabbage mosaic virus. Larson & Walker 1939 (in part) fide Smith *in litt.*
Broccoli mosaic virus. Caldwell & Prentice 1942.
Strains or suspected strains.
Cauliflower virosis virus. Moericke & Winter 1940.
Cabbage virus B. Walker, LeBeau, & Pound 1945.

Celery Mosaic. Smith *Tb.* 204; Holmes *Hb.* 67.
Celery mosaic virus. Holmes *Hb.*
Celery western mosaic virus. Severin & Freitag 1935, 1938.
Apium virus 1. Smith *Tb.*
Marmor umbelliferarum Holmes *Hb.*

Celery Southern Mosaic, caused by Cucumber mosaic virus (str.).

Celery Aster Yellows, caused by Aster yellows virus (str.).

Cherry Banded Chlorosis. Hildebrand *et al. Hb.* 50; Brierley *P.D.R.S. 158*, 189.
(**Flowering**) **Cherry banded chlorosis virus.** Zeller & Milbrath 1942.
Marmor pallidolimbatus Zeller & Milbrath 1942.
Prunus virus 10. Zeller & Milbrath 1942.

Cherry Mottle Leaf. Hildebrand *et al. Hb.* 42.
Cherry mottle leaf virus. Zeller 1934, Reeves 1941.
Prunus virus 7. Zeller & Evans 1941.
Marmor cerasae Zeller & Evans 1941.

Cherry Rough Bark. Hildebrand *et al. Hb.* 42; Brierley *P.D.R.S. 150*, 467.
(**Flowering**) **Cherry rough bark virus.** Milbrath & Zeller 1942.
Prunus virus 9. Milbrath & Zeller 1942.
Rimocortius kwanzani Milbrath & Zeller 1942.

Cherry Rusty Mottle. Hildebrand *et al. Hb.* 46.
Cherry rusty mottle virus.
Sweet cherry rusty mottle virus. Reeves 1940, 1941.
Marmor rubiginosum Reeves 1940, 1941.

Cherry Vein-clearing. Hildebrand *et al. Hb.* 47.
Cherry vein-clearing virus. Zeller & Evans 1941.
Prunus virus 8. Zeller & Evans 1941.
Marmor nerviclarens Zeller & Evans 1941.

Cherry Yellows. Keitt & Clayton, *Phytopathology*, xxxiii; 449–468, 1943.
Cherry yellows virus. Hildebrand 1942, Keitt & Clayton 1943.
[See Keitt & Clayton for possible synonyms.]

Citrus Infectious Mottling. Fawcett, *Citrus diseases and their control* (Ed. 2), 356, 1936; *Phytopathology*, xxxi, 356, 1941.
Citrus infectious mottling virus. Fawcett 1936.
Variegatura infettiva di citrus virus. Petri 1931.
Citrivir italicum Fawcett 1941.

Citrus Psorosis. Fawcett, *Citrus diseases and their control* (Ed. 2), 188, 1936; Fawcett & Bitancourt, *Phytopathology*, xxxiii, 837–864, 1943.
Citrus psorosis virus. [Swingle & Weber 1896] Fawcett 1934.
Citrivir psorosis Fawcett 1941.
Strains or suspected strains.
Citrus psorosis A virus.
Citrivir psorosis var. *vulgare* Fawcett 1941.
Citrus psorosis B virus.
Citrivir psorosis var. *annulatum* Fawcett 1941.
Concave gum psorosis virus. Fawcett & Bitancourt 1943.
Citrivir psorosis var. *concavum* Fawcett & Bitancourt 1943.
Blind pocket psorosis virus. Fawcett & Bitancourt 1943.
Citrivir psorosis var. *alveatum* Fawcett & Bitancourt 1943.
Crinkly leaf psorosis virus. Fawcett & Bitancourt 1943.

Clover Big Vein. Black, *Proc. Amer. phil. Soc.*, lxxxviii, 132–144, 1944; *Amer. J. Bot.*, xxxii, 407–415, 1945.
Clover big vein virus. Black 1944.
Aureogenus magninerva Black 1944.
Wound-tumour virus. Black 1945.

Clover Club Leaf. Black, *Proc. Amer. phil. Soc.*, lxxxviii, 132–144, 1944.
Clover club leaf virus. Black 1944.
Aureogenus clavifolium Black 1944.

Clover Mosaic, Alsike. Weiss *P.D.R.S. 154*, 62.
Alsike clover mosaic virus. Zaumeyer & Wade 1935.
Alsike clover virus 1. Zaumeyer & Wade 1936.
Alsike virus 1. Zaumeyer & Wade 1938.
Trifolium virus 3. Zaumeyer in Weiss 1939.
Alsike clover mosaic virus 1. Zaumeyer 1940.
Strains or suspected strains.
Alsike clover mosaic virus 2. Zaumeyer.
Trifolium virus 3A. Zaumeyer in Weiss 1939.

Clover Vein Mosaic. Holmes *Hb.* 93.
Clover vein mosaic virus.
Red clover vein mosaic virus. Osborn 1937.
? Pea-streak virus 1. Zaumeyer & Wade 1937.
Marmor trifolii Holmes *Hb.*
Trifolium virus 2. Weiss 1939.

Corn, see Maize.

Cotton Leaf Curl. Smith *Tb.* 90; Holmes *Hb.* 116.
Cotton leaf curl virus. Bailey 1934, Kirkpatrick 1931.
Cotton leaf-crinkle virus. Kirkpatrick 1930.
Gossypium virus 1. Smith *Tb.*
Ruga gossypii Holmes *Hb.*

Cowpea Mosaic, caused by Cucumber mosaic virus (str.).

Cranberry False Blossom. Smith *Tb.* 205; Holmes *Hb.* 10.
Cranberry false blossom virus. [Shear 1919] Dobroscky 1929, 1931.
Wisconsin false-blossom [virus]. fide Dobroscky.

Vaccinium virus 1. Smith *Tb.*
Chlorogenus vaccinii Holmes *Hb.*

Cucumber Mosaic. Smith *Tb.* 52; Holmes *Hb.* 31–40 (excluding var. *upsilon*).
Cucumber mosaic virus. Selby 1910; Doolittle 1916, 1920.
Calico or mosaic of cucumber and melon [virus]. Stone 1910.
Cucumber wart disease [virus]. Ruggles & Stakman 1911.
Cucumber white pickle mosaic virus. Gilbert 1916.
Spinach blight virus. McClintock 1918, I. F. Storey 1939 fide Smith *Tb.*
Cucumber virus 1. J. Johnson 1927.
Spinach mosaic virus. L. B. Smith 1920.
Tobacco puff virus. Valleau & E. M. Johnson 1928 fide E. M. Johnson 1930.
Tomato fern leaf virus (in part) fide Mogendorff 1930.
Cucumber mosaic virus type 1. E. M. Johnson 1930.
White pickle virus. Porter 1931.
Cucumber yellow mosaic virus. Ainsworth 1934.
Tomato narrow leaf virus. Chamberlain 1934.
Cucumber yellow-mottle mosaic virus. Ainsworth 1935.
Ordinary cucumber mosaic virus. Hoggan 1935.
Lupinbräune virus. Köhler 1935.
Cucumis virus 1. Smith *Tb.*
Marmor cucumeris var. *vulgare* Holmes *Hb.*
Cucumber mosaic virus, typical strain. Holmes *Hb.*
Abaca mosaic virus. Calinisan 1939, Celino 1940.
Murialba cucumeris (Holmes) Valleau 1940.
Cucumber mosaic virus, strain 1. Phillips 1942.
Strains or suspected strains.
? Abaca mosaic virus. Porter 1930.
Banana heart rot and infectious chlorosis virus. Magee 1930, Wardlaw & McGuire 1933.
Banana mosaic virus. Ogilvie 1927 fide Wardlaw 1935.
Banana virus 2. J. Johnson in Smith *Tb.*
Musa virus 2. Smith *Tb.*
Bettendorf mosaic virus. Porter 1930.
Cucumber virus 2. Porter 1931.
? Cucumber mild mosaic virus. Hoggan 1935 fide Hoggan 1935.
Cucumis virus 1D. Smith *Tb.*
Bottle gourd (*Lagenaria vulgaris*) mosaic virus. Vasudeva & Lal 1943.
Cucumis virus 3. Vasudeva & Lal 1943.
Cantaloupe mosaic virus. Middleton & Whitaker 1942.
Celery southern mosaic virus. Wellman 1935.
Celery mosaic virus. Poole 1921, Doolittle 1931.
Celery virus 1. Wellman 1935.
Marmor cucumeris var. *commelinae* Holmes *Hb.*
Cucumber-mosaic virus, southern celery-mosaic strain. Holmes *Hb.*
Commelina mosaic virus. Kunkel 1921, Carter 1937.
Commelina mosaic virus yellow and green strains. Price 1941.
Cowpea mosaic virus. Gardner 1927, McLean 1941.
? Cucumber mosaic virus, strain Y. Price 1934 fide McLean 1941.
Cucumis virus 10. Smith *Tb.*
Marmor cucumeris var. *vignae* Holmes *Hb.*
Cucumber-mosaic virus, cowpea-mottling strain. Holmes *Hb.*

Cucumber mosaic virus, strain 5 (yellow cucumber mosaic virus). Price 1934.
Cucumis virus 1A. Smith *Tb.*
Cucumber mosaic virus, strain 6. Price 1934.
Cucumeris virus 1B. Smith *Tb.*
Marmor cucumeris var. *judicis* Holmes *Hb.*
Cucumber mosaic virus, indicator strain. Holmes *Hb.*
Cucumber mosaic virus, type 2. E. M. Johnson 1930.
? Yellow cucumber mosaic virus. Hoggan 1935 fide Hoggan 1935.
Cucumber mosaic virus, type 3. E. M. Johnson 1930.
Cucumber mosaic virus, strain 3. Phillips 1942 fide Phillips 1942.
Cucurbit yellow mosaic virus. McKinney 1929.
Yellow cucumber mosaic virus. Hoggan 1935.
Delphinium ring spot virus. Severin & Dickson 1942.
Delphinium stunt disease virus. Burnett 1934 fide Valleau 1940.
Delphinium witch's broom disease virus.
Delphinium virus 2. Smith *Tb.*
Lily mosaic virus. Price 1937, Kawamura 1938, Ainsworth 1938.
Lily virus, cucumber mosaic strain. Brierley 1939.
Marmor cucumeris var. *lilii* Holmes *Hb.*
Cucumber mosaic virus, lily-mosaic strain. Holmes *Hb.*
Lima bean mosaic virus. Harter 1936.
Marmor cucumeris var. *phaseoli* Holmes *Hb.*
Cucumber mosaic virus, lima-bean mosaic strain. Holmes *Hb.*
Nicotiana viscosa mosaic virus. Allard 1916.
Primula obconica virus. K. M. Smith 1935.
Tobacco bleaching mosaic virus. J. Johnson 1927 fide Valleau 1940.
Tobacco virus 8. J. Johnson 1927.
Nicotiana virus 4. Smith *Tb.*
Tobacco mild mosaic virus. J. Johnson 1927 fide Valleau 1940.
Tobacco virus 3. J. Johnson 1927.
Nicotiana virus 3. Smith *Tb.*

Cucumber Green-mottle Mosaic. Smith *Tb.* 83; Holmes *Hb.* 27.
Cucumber green-mottle mosaic virus. Ainsworth 1935.
Cucumber mild or ordinary mosaic virus. Bewley 1923.
Cucumber virus 3. Ainsworth 1935.
Cucumis virus 2. Smith *Tb.*
Marmor astrictum var. *chlorogenum* Holmes *Hb.*
Cucurbit-mosaic virus, green-mottling strain. Holmes *Hb.*
Musivum astrictum Valleau 1940.
Strain or suspected strain.
Cucumber yellow mosaic virus. Ainsworth 1935.
Cucumber aucuba mosaic virus. Bewley 1923.
Cucumber virus 4. Ainsworth 1935.
Cucumis virus 2A. Smith *Tb.*
Marmor astrictum var. *aucuba* Holmes *Hb.*
Cucurbit mosaic virus, aucuba-mosaic strain. Holmes *Hb.*

Cucumber Yellow Mosaic, caused by Cucumber green-mottle mosaic virus (str.).

Currant Reversion. Smith *Tb.* 97; Holmes *Hb.* 112.
Currant reversion virus. [Lees 1920, Amos & Hatton 1926] Amos *et al.* 1928.
Ribes virus 1. Smith *Tb.*

Acrogenus ribis Holmes *Hb.*
Black-currant reversion disease virus.

Dahlia Mosaic. Smith *Tb.* 208; Holmes *Hb.* 85; Brierley *P.D.R.S. 150*, 428; *ibid.* 158, 176.
Dahlia mosaic virus. Brierley 1933.
Dahlia yellows and mosaic [viruses]. Howe 1922 fide Smith *Tb.*
Dahlia stunt or dwarf [virus]. Howe 1923 fide Smith *Tb.*
Dahlia runting [virus]. Conners 1926 fide Smith *Tb.*
Dahlia leaf curl and rosette [virus]. Martin 1929 fide Smith *Tb.*
Dahlia virus 1. Smith *Tb.*
Marmor dahliae Holmes *Hb.*

Dahlia Oak Leaf. Smith *Tb.* 214.
Dahlia oakleaf virus. Brierley 1933.
Dahlia virus 3. Smith *Tb.*

Dahlia Ring Spot. Smith *Tb.* 213.
Dahlia ringspot virus. Brierley 1933.
Dahlia virus 2. Smith *Tb.*
Strain or suspected strain.
Dahlia yellow ringspot virus. Brierley 1933.
Dahlia virus 2B. J. Johnson in Smith *Tb.*
Dahlia virus 2A. Smith *Tb.*

Dandelion Yellow Mosaic. Kassanis, *Nature, Lond.*, cliv, 16, 1944.
Dandelion yellow mosaic virus. Kassanis 1944.

Dock Mosaic. Smith *Tb.* 598; Brierley *P.D.R.S. 150*, 473.
Dock mosaic virus. Grainger & Cockerham 1930.
Dock infectious chlorosis (virus). Grainger 1928.
Rumex virus 1. Smith *Tb.*
Rumex obtusifolius virus. Fernow 1925 fide Brierley 1944.

Dodder Latent Mosaic. Bennett, *Phytopathology*, xxxiv, 77–91, 1944; Brierley *P.D.R.S. 158*, 175.
Dodder latent mosaic virus. Bennett 1944.
Marmor secretum Bennett 1944.
Cuscuta virus 1. Bennett 1944.
Cuscutavir secretum Bennett 1944.
Cuscuta latent virus. Brierley 1945.

Euonymus Variegation. Holmes *Hb.* 51; Brierley *P.D.R.S. 150*, 436.
Euonymus variegation virus. Brierley 1944.
Euonymus infectious chlorosis [virus]. Baur 1908.
Marmor euonymi Holmes *Hb.*
Euonymus-mosaic virus. Holmes.

Fig Mosaic. Smith *Tb.* 192.
Fig mosaic virus. Condit & Horne 1933.
Ficus virus 1. Smith *Tb.*
Ficivir caricae Condit & Horne 1941.

Groundnut Rosette. Smith *Tb.* 185; Holmes *Hb.* 67; Weiss *P.D.R.S. 154*, 35.
Groundnut rosette virus. Storey & Bottomley 1928.
Groundnut Kräuselkrankheit [virus]. Zimmerman 1907.

Groundnut lèpre (gana) [virus]. Trochain 1931.
Arachis virus 1. Smith *Tb.*
Marmor arachidis Holmes *Hb.*
Peanut rosette virus. Holmes *Hb.*

Strain or suspected strains.
Chlorosis rosette, green rosette, and type no. 3 viruses. Hayes 1932.

Henbane Mosaic. Smith *Tb.* 331; Brierley *P.D.R.S. 150*, 443.
Henbane mosaic virus.
Hyoscyamus virus 1. Smith *Tb.*
Hy. III virus. Hamilton 1932.
Hyoscyamus mosaic virus Brierley 1944.

Holodiscus Witches' Broom. Smith *Tb.* 127; Holmes *Hb.* 127; Brierley *P.D.R.S. 150*, 439.
Holodiscus witches' broom virus.
Ocean spray witches' broom virus. Zeller 1931.
Holodiscus virus 1. Smith *Tb.*
Nanus holodisci Holmes *Hb.*

Hop Chlorotic Disease. Smith *Tb.* 194.
Hop chlorotic disease virus. Salmon & Ware 1930.
Humulus virus 3. Smith *Tb.*

Strain or suspected strain.
Fuggles hop mosaic disease virus. Salmon & Ware 1936.
Humulus virus 4. Smith *Tb.*
Chlorogenus humuli Holmes *Hb.*

Hop Nettlehead. Smith *Tb.* 194; Holmes *Hb.* 15.
Hop nettlehead virus. [Duffield 1925].
Silly-hill virus fide Holmes *Hb.*
'Eelworm' disease [virus] fide Smith *Tb.*
? Hop infectious sterility virus. Blattny 1935.
Humulus virus 2. Smith *Tb.*
? Hop Kräuselkrankheit or curl disease [virus].

Iris Mosaic. Smith *Tb.* 420; Holmes *Hb.* 55; Brierley *P.D.R.S. 150*, 445.
Iris mosaic virus. Brierley & McWhorter 1936.
Iris stripe virus. Brierley & McWhorter 1934.
Iris virus 1. Smith *Tb.*
Marmor iridis Holmes *Hb.*

Laburnum Variegation. Smith *Tb.* 555; Holmes *Hb.* 51; Brierley *P.D.R.S. 150*, 447.
Laburnum variegation virus. Brierley 1944.
Laburnum infectious chlorosis [virus]. Baur 1907.
Marmor laburni Holmes *Hb.*
Laburnum mosaic virus. Holmes *Hb.*

Lettuce Mosaic. Holmes *Hb.* 84.
Lettuce mosaic virus. Jagger 1921, Ainsworth & Ogilvie 1939.
Lactuca virus 1. Smith *Tb.*
Marmor lactucae Holmes *Hb.*

Lily Necrotic Fleck, caused by Lily symptomless virus+Cucumber mosaic virus.

Lily Symptomless Virus Disease. Brierley & F. F. Smith, *Phytopathology*, xxxiv, 529–555, 1944.

Lily symptomless virus. Brierley & Smith 1944.

Adelonosus lilii Brierley & Smith 1944.

Lily Rosette. Smith *Tb.* 415; Brierley *P.D.R.S. 150*, 449; *ibid. 158*, 180.

Lily rosette virus. Ogilvie 1928.

Lily yellow-flat virus. Ogilvie 1928.

Lilium virus 1. Smith *Tb.*

Lucerne Dwarf. Smith *Tb.* 181; Weiss *P.D.R.S. 154*, 36.

Lucerne dwarf virus.

Alfalfa dwarf virus. Weimer [1931] 1936.

Medicago virus 3. Smith *Tb.*

Lucerne Mosaic. Smith *Tb.* 179; Holmes *Hb.* 91; Weiss *P.D.R.S. 154*, 38.

Lucerne mosaic virus.

Alfalfa mosaic virus. Weimer 1931.

Alfalfa ringspot virus. Young 1934.

Alfalfa virus 1. Pierce 1934.

Medicago virus 1. Smith *Tb.*

Alfalfa mosaic virus 1. Zaumeyer 1938.

Alfalfa virus 2. Pierce fide Zaumeyer 1938.

Medicago virus 2. Smith *Tb.*

Marmor medicaginis Holmes *Tb.*

Marmor medicaginis Holmes var. *typicum* Black & Price 1940.

Chilli mosaic virus Kovačevsky 1942 fide Kovačevsky 1942.

Medicago virus 2 Smith var. *typicum* Black & Price, Kovačevsky 1942.

Strains or suspected strains.

Alfalfa mosaic virus 1A. Zaumeyer 1938.

Alfalfa mosaic virus 1B. Zaumeyer 1938.

Potato calico virus. Hungerford 1922, Porter 1931.

Solanum virus 10. Smith *Tb.*

Marmor medicaginis Holmes var. *solani*. Black & Price 1940.

Lucerne Witches' Broom. Smith *Tb.* 182; Holmes *Hb.* 14.

Alfalfa witches' broom (Amer.).

Lucerne witches' broom virus. Edwards 1936.

Spindle shoot virus. Edwards 1936.

Mistletoe virus. Edwards 1936.

Kurrajong virus. Edwards 1936.

Bunchy top virus. Edwards 1936.

Medicago virus 4. Smith *Tb.*

Chlorogenus medicaginis Holmes *Hb.*

Lupin Sore Shin, caused by Pea mosaic virus.

Maize Mosaic. Smith *Tb.* 439; Holmes *Hb.* 59.

Corn mosaic (Amer.).

Maize mosaic virus. Kunkel 1923.

Corn stripe virus. Stahl 1927.

Corn leaf stripe virus. Stahl 1927.

Yellow stripe disease virus. Kunkel 1922 fide Holmes *Hb.*

Corn virus 1. J. Johnson in Smith *Tb.*

Zea virus 1. Smith *Tb.*

Marmor zeae Holmes *Hb.*

Maize Streak. Smith *Tb.* 441; Holmes *Hb.* 56.
Maize streak virus. Storey 1925.
Corn virus 2. J. Johnson in Smith *Tb.*
Zea virus 2. Smith *Tb.* (in part) fide Holmes *Hb.*
Marmor maidis var. *typicum* Holmes *Hb.*
Maize-streak virus, typical strain. Holmes *Hb.*
Fractilinea maidis (Holmes) McKinney 1944.
Strains or suspected strains.
Maize-streak virus, cane-streak strain. Holmes *Hb.*
Marmor maidis var. *sacchari* Holmes *Hb.*
Zea virus 2. Smith *Tb.* (in part).
Sugar cane streak disease virus. Storey 1925.
Maize-streak virus, mottle strain. Holmes *Hb.*
Marmor maidis var. *mite* Holmes *Hb.*
Zea virus 3. Smith *Tb.*
Maize mottle virus. Storey 1937.

Maize Stripe. H. H. Storey, *E. Afr. agric. J.*, i, 333–7, 1936.
Maize stripe virus. Storey 1936.

Maize Wallaby Ear. McKinney, *J. Wash. Acad. Sci.*, xxxiv, 328, 1944.
Maize wallaby-ear virus. Schindler 1942.
Galla zeae McKinney 1944.

Mountain Ash Variegation. Smith *Tb.* 149.
Mountain ash variegation virus.
Infectious chlorosis [virus]. Baur 1907, Hertsch 1930.
Pyrus variegation virus. Atanasoff 1935.
Pyrus virus 1. Smith *Tb.*

Narcissus Mosaic. Brierley *P.D.R.S. 150*, 453, 455.
Narcissus Stripe.
Narcissus mosaic virus. McWhorter & Weiss 1932, Haasis 1939.
Yellow stripe [virus]. Darlington 1908 fide Haasis 1939.
Gray disease virus. Griffiths & Juenemann 1919, Griffiths 1924.

Oat Pseudo-rosette. McKinney, *J. Wash. Acad. Sci.*, xxxiv, 327–8, 1944.
Oat pseudo-rosette virus. Grebennikov 1941.
Oat 'zakuklivanie' (pupation disease) virus. [Donchenko, 1929, Sukhov & Vovk 1937] Sukhov & Sukhova 1940.
Siberian oat mosaic virus. McKinney 1944.
Fractilinea avenae McKinney 1944.

Onion Yellow Dwarf. Smith *Tb.* 418; Holmes *Hb.* 66.
Onion yellow dwarf virus. Melhus *et al.* 1929; Drake, Harris, & Tate 1932.
Onion brittle [virus]. Clinton 1905 fide M. T. Cooke 1934.
? Onion Rotzkrankheit virus. Bremer [1929] 1937.
Zwiebelrotz virus. Molz & Müller 1926 fide Bremer 1937.
Onion crinkles virus. fide Smith *Tb.*
Allium virus 1. Smith *Tb.*
Marmor cepae Holmes *Hb.*

Ornithogalum Mosaic. Smith (F. F.) & Brierley, *Phytopathology*, xxxiv, 497–503, 1944.
Ornithogalum mosaic virus. Smith & Brierley 1944.
Bulb-plant-mosaic [virus]. Atanasoff 1928 (in part).
Marmor scillearum Smith & Brierley 1944.

Passion Fruit Woodiness. Smith *Tb.* 50; Holmes *Hb.* 77; Brierley *P.D.R.S. 150*, 459.

Passion fruit woodiness virus. [Cobb 1901.]
Passion fruit bullet disease virus. Noble 1928.
Passiflora virus 1. Smith *Tb.*
Marmor passiflorae Holmes *Hb.*

Pea Enation Mosaic. Smith *Tb.* 165; Holmes *Hb.* 90; Weiss *P.D.R.S. 154*, 53.

Pea enation mosaic virus. Pierce 1935.
Pea mosaic virus. Osborn 1935.
Pea virus 1. Pierce 1935, Stubbs 1937.
Pisum virus 1. Smith *Tb.*
Marmor pisi Holmes *Hb.*

Pea Mosaic. Smith *Tb.* 168; Holmes *Hb.* 89; Weiss *P.D.R.S. 154*, 51.

Pea mosaic virus. Doolittle & Jones 1925, Chamberlain 1936, 1937.
Garden pea mosaic virus. Dickson 1921.
Lupin sore shin virus. Neill *et al.* 1934 fide Chamberlain 1935.
Common pea mosaic virus. Pierce 1935.
Pea virus 3. Pierce 1935. Murphy & Pierce 1937 fide Smith *Tb.*
Pea mosaic virus 1. Zaumeyer & Wade 1935 fide Smith *Tb.*
Pea virus 2. Osborn 1937.
Pisum virus 2. Smith *Tb.*
Marmor leguminosarum Holmes *Hb.*

Strains or suspected strains.
Pea marble mosaic virus. Stubbs 1936, 1937.
Pea virus 2A. Stubbs.
Pisum virus 2A. Smith *Tb.*
Pea speckle mosaic virus. Stubbs 1936, 1937.
Pea virus 2B. Stubbs.
Pisum virus 2B. Smith *Tb.*
Pea mild mosaic virus. Stubbs 1936, 1937.
Pea virus 2C. Stubbs.
Pisum virus 2C. Smith *Tb.*

Pea Mottle. Weiss *P.D.R.S. 154*, 55.

Pea mottle virus. F. Johnson 1942.
Marmor efficiens F. Johnson 1942.
White clover mosaic virus. Pierce 1935 (in part) fide F. Johnson.
White clover virus 1. Pierce 1935.
Trifolium virus 1. Weiss 1939.
Broad bean mosaic virus. Pierce 1935 fide F. Johnson.
Severe pea mosaic virus. F. Johnson & Jones 1937 fide J. Johnson.
? Pea virus 2. Zaumeyer & Wade 1935.

Pea Streak. Weiss *P.D.R.S. 154*, 58, 59.

Pea streak virus. Zaumeyer & Wade, *J. agric. Res.*, lvi, 747, 1938.
Pea streak virus 1. Zaumeyer 1938.
Pisum virus 5. Weiss 1939.
Pea American streak virus. Weiss 1944.
Pea streak virus. Chamberlain, *N.Z. J. Sci. Tech.*, A, xx, 365, 1939.
Pisum virus 3. Chamberlain 1939.
Pea New Zealand streak virus. Weiss 1944.

Pea Wilt. Weiss *P.D.R.S. 154*, 57.
Pea wilt virus. F. Johnson 1942.
Marmor repens F. Johnson 1942.
White clover mosaic virus. Pierce 1935 (in part) fide F. Johnson.
White clover virus 1. Pierce 1935.
Trifolium virus 1. Weiss 1939.
Broad-bean local-lesion virus. Pierce 1935 fide F. Johnson.

Peach Asteroid Spot. Holmes *Hb.* 83; Hildebrand *et al. Hb.* 25.
Peach asteroid spot virus. Cochran & C. O. Smith 1938.
Marmor astri Holmes *Hb.*

Peach Line-pattern Virosis. Hildebrand *et al. Hb.* 35.
Peach line-pattern virosis virus. Cation 1940.
Marmor lineopictum Cation 1941.
? Plum and peach virus. Valleau 1932.
? Vacaville plum mosaic virus. Thomas & Rawlins 1939.

Peach Little Peach, caused by Peach yellows virus (str.).

Peach Mosaic. Smith *Tb.* 142; Holmes *Hb.* 81; Hildebrand *et al. Hb.* 17.
Peach mosaic virus. Hutchins 1933.
Peach virus 6. J. Johnson in Smith *Tb.*
Prunus virus 5. Smith *Tb.*
Marmor persicae Holmes *Hb.*
Flavimacula persicae (Holmes) McKinney 1944.

Peach Phony. Smith *Tb.* 136; Holmes *Hb.* 126; Hildebrand *et al. Hb.* 15.
Phony peach virus. [Neal 1921] Hutchins 1930.
Peach virus 4. J. Johnson in Smith *Tb.*
Prunus virus 3. Smith *Tb.*
Nanus mirabilis Holmes *Hb.*

Peach Red Suture. Smith *Tb.* 141; Hildebrand *et al. Hb.* 13 [? caused by a strain of Peach yellows virus fide Holmes].
Peach red-suture virus. Bennett 1926, Cation 1932.
Peach virus 5. J. Johnson in Smith *Tb.*
Prunus virus 4. Smith *Tb.*

Peach Rosette. Smith *Tb.* 134; Holmes *Hb.* 125; Hildebrand *et al. Hb.* 10.
Peach rosette virus. [E. F. Smith 1891] McClintock 1923.
Peach virus 2. J. Johnson in Smith *Tb.*
Prunus virus 2. Smith *Tb.*
Nanus rosettae Holmes *Hb.*

Peach Rosette Mosaic. Hildebrand *et al. Hb.* 19.
Peach rosette mosaic virus. Cation 1933, Hildebrand 1941.
Michigan rosette virus. Cation 1933.

Peach Wart. Blodgett, *Phytopathology*, xxxiii, 21–32, 1943.
Peach wart virus. Blodgett 1943.
Peach blister [virus]. Blodgett 1939.
Galla verruca Blodgett 1943.

Peach Yellow Bud Mosaic. Thomas, Scott, & Wilson, *Phytopathology*, xxxiv, 658–661, 1944.
Peach yellow bud mosaic virus. Thomas *et al.* 1944.

Winters peach mosaic virus. Thomas & Rawlins 1939.
Inops consilii Thomas *et al.* 1944.

Peach Yellows. Smith *Tb.* 128; Holmes *Hb.* 5; Hildebrand *et al. Hb.* 7, 11.
Peach yellows virus. [E. F. Smith 1888] Kunkel 1933.
Peach virus 1. J. Johnson in Smith *Tb.*
Prunus virus 1. Smith *Tb.*
Chlorogenus persicae var. *vulgare* Holmes *Hb.*
Peach-yellows virus, typical strain. Holmes *Hb.*
Strain or suspected strain.
Peach little peach virus. [E. F. Smith 1891] Bennett 1926.
Peach virus 3. J. Johnson in Smith *Tb.*
Prunus virus 1A. Smith *Tb.*
Chlorogenus persicae var. *micropersica* Holmes *Hb.*
Peach-yellows virus, little-peach strain. Holmes *Hb.*

Peach X-Disease. Holmes *Hb.* 82; Hildebrand *et al. Hb.* 21, 29, 39.
Peach X-disease virus. Stoddard 1938.
Peach yellow-red virosis virus. Hildebrand & Palmiter 1938, 1943.
Marmor lacerans. Holmes *Hb.*
Carpophthora lacerans. (H.) McKinney 1944.
Sweet cherry buckskin virus. Rawlins & Horne 1931, Rawlins & Thomas 1941 fide Rawlins & Thomas 1941.
Peach leaf casting yellows virus. Thomas, Rawlins, & Parker 1940 fide Thomas *et al.* 1940.
Strain or suspected strain.
Peach western X-disease virus. Zeller & Evans 1941.

Peanut, see Groundnut.

Pear Stony Pit. Holmes *Hb.* 76.
Pear stony pit virus. Kienholz 1939.
Marmor pyri Holmes *Hb.*

Pelargonium Leaf Curl. Smith *Tb.* 48; Brierley *P.D.R.S. 150*, 460.
Pelargonium leaf curl virus. Pape 1927.
Pelargonium virus. Verplancke 1932.
Pelargonium virus. Blattný 1933.
Pelargonium virus 1. Smith *Tb.*
Geranium leaf curl virus. Berkeley 1938.
Geranium crinkle virus. L. K. Jones 1938.
Geranium mosaic virus. L. K. Jones 1940.

Peony Ring Spot. Smith *Tb.* 5; Brierley *P.D.R.S. 150*, 458.
Peony ring spot virus. Green 1935, Dufrénoy 1934.
Peony infectious chlorosis virus. Fukushi 1932 fide Brierley 1944.
Paeonia virus 1. Smith *Tb.*
Peony mosaic virus. Brierley 1944.

Plantago Mosaic, caused by Tobacco mosaic virus (str.).

Plum Dwarf. Smith *Tb.* 148; Holmes *Hb.* 128; Hildebrand *et al. Hb.* 69.
Plum dwarf virus.
Prune dwarf virus. Thomas & Hildebrand 1936.
Prunus virus 6. Smith *Tb.*
Nanus pruni Holmes *Hb.*

Potato Apical Leaf Roll. Smith *Tb.* 389.
Potato apical leaf roll virus. Schultz & Bonde 1929.
Potato yellow top virus. Folsom 1926 fide Smith *Tb.*
Solanum virus 17. Smith *Tb.*

Potato Aucuba Mosaic. Smith *Tb.* 371, 367; Holmes *Hb.* 49; Weiss *P.D.R.S. 155*, 102, 103, 105.
Potato aucuba mosaic virus. Quanjer 1922.
Potato non-infectious chlorosis virus. Murphy fide Smith *Tb.*
Potato virus G. Clinch, Loughnane, & Murphy 1936.
Solanum virus 9. Smith *Tb.*
Marmor aucuba Holmes *Hb.*
Potato pseudo-net-necrosis virus. Quanjer 1929 fide Holmes *Hb.*
Potato phloem-parenchyma necrosis virus. Quanjer 1934.
Potato tuber blotch virus. Loughnane & Clinch 1934.
Potato streak virus. Koch & Johnson 1935 fide Loughnane & Clinch 1935.
Monocraat virus. Loughnane & Clinch 1935, Dykstra 1939.
Potato virus F. Clinch *et al.* 1936.
Solanum virus 8. Smith *Tb.*
Canada streak virus. Dykstra 1939 fide Holmes *Hb.*
Marmor aucuba Holmes var. *canadense* Black & Price 1940.

Potato Calico, caused by Lucerne mosaic virus (str.).

Potato Crinkle, caused by Potato virus X+Potato virus A.
American Mild Mosaic and Crinkle Mosaic.

Potato Leaf Drop Streak, caused by Potato virus Y.

Potato Leaf Roll. Smith *Tb.* 380; Holmes *Hb.* 120; Weiss *P.D.R.S. 155*, 106.
Potato leaf roll virus. [Appel 1911.]
Potato Kräuselkrankheit [virus]. Appel 1907.
Potato leaf curl [virus]. Pethybridge 1911.
Potato phloem necrosis virus. Quanjer 1913.
Potato virus 1. J. Johnson in Smith *Tb.*
Solanum virus 14. Smith *Tb.*
Corium solani Holmes *Hb.*

Potato Leaf-rolling Mosaic. Smith *Tb.* 375; Weiss *P.D.R.S. 155*, 100.
Potato leaf-rolling mosaic virus. [Schultz & Folsom 1923] Folsom 1926, J. Johnson 1929.
Potato virus 7. J. Johnson in Smith *Tb.*
Solanum virus 11. Smith *Tb.*
Potato virus E. Dykstra 1939.
? Potato virus K. Köhler 1941, 1942 fide Köhler.
Potato rolling mosaic virus. Köhler 1935.

Potato Mottle. Smith *Tb.* 341; Holmes *Hb.* 42; Weiss *P.D.R.S. 155*, 84.
Potato Mild Mosaic (Brit.).
Potato Latent Mosaic (Amer.).
Potato virus X. K. M. Smith 1931.
Potato common mosaic virus. Quanjer 1923.
Potato mosaic B virus. Fernow 1925.
Potato mottle virus. J. Johnson 1925.

Latent potato virus. Schultz 1925.
Tobacco ring spot virus. J. Johnson 1927 fide Smith *Tb.*
Tobacco virus 5. J. Johnson 1927.
Healthy potato virus. E. M. Johnson 1930.
Potato virulent latent virus. Burnett & Jones 1931.
Potato virus X, S strain. Salaman 1930.
Potato ring spot virus. J. Johnson 1925, Koch 1932.
Marmor dubium var. *annulus* Holmes *Hb.*
Potato-mottle virus, potato-ringspot strain. Holmes *Hb.*
Interveinal mosaic virus. Quanjer fide Smith *Tb.*
Potato top-necrosis virus. Quanjer 1931.
Potato acronecrosis virus. Quanjer 1931.
Potato simple mosaic virus. Murphy 1932.
Hy. IV virus. Hamilton 1932.
Potato virus 16. J. Johnson in Smith *Tb.*
Solanum virus 1. Smith *Tb.*
Marmor dubium var. *vulgare* Holmes *Hb.*
Potato-mottle virus, typical strain. Holmes *Hb.*
Potato mild mosaic virus. Samuel 1943.

Strains or suspected strains.
Masked virus of Aucklander short-top potatoes. Chamberlain 1941.
Potato interveinal necrosis virus. Salaman 1938.
Potato virus XN. Salaman 1938.
Potato-mottle virus, masked-mottle strain. Holmes *Hb.*
Marmor dubium var. *obscurum* Holmes *Hb.*
Potato virus X, strains XH, XG, XL, and XS. Salaman 1938.
Potato X-1 strain. Böhme 1933.
Potato yellow-mottle virus. Putnam 1937.
Potato yellow-mottle X-type virus. Bald & Pugsley 1941.
President streak virus. Murphy 1927 fide Bawden 1943.
Potato foliar necrosis virus. Bawden 1934.
Potato virus D. Bawden 1934.
Solanum virus 6. Smith *Tb.*
Potato virus XD. Salaman 1938.
Tomato necrosis virus. Chamberlain 1940.
Up-to-Date streak virus. Murphy & McKay 1926 fide Clinch 1942.
Potato virus B. Bawden in Smith 1933, Bawden 1936.
Solanum virus 4. Smith *Tb.*, Dennis 1939.

Potato Net Necrosis, caused by Potato leaf roll virus+Potato virus A.

Potato Paracrinkle. Smith *Tb.* 365; Holmes *Hb.* 48; Weiss *P.D.R.S. 155*, 99.
Potato paracrinkle virus. Salaman and LePelley 1930.
Potato virus E. Bawden in Smith 1933.
Solanum virus 7. Smith *Tb.*
Marmor angliae Holmes *Hb.*

Potato Purple Top Wilt, caused by Aster yellows virus.

Potato Rugose Mosaic. Smith *Tb.* 348; Holmes *Hb.* 33; Weiss *P.D.R.S. 155*, 88, 92.
Potato Severe Mosaic (Brit.).

Potato virus Y. Smith 1931.
Streak virus. Orton 1920 fide Smith *Tb.*
Leaf-drop streak virus. Murphy 1921 fide Smith *Tb.*
Stipple-streak virus. Atanasoff 1922 fide Smith *Tb.*
(Tobacco and potato) veinbanding virus. E. M. Johnson 1930, Valleau & Johnson 1930 fide Smith *Tb.*
Tobacco vein margin virus. Valleau & Johnson 1928.
Acropetal necrosis virus. Quanjer 1931.
Hy. II virus. Hamilton 1932 fide Smith 1933.
Potato virus 20. J. Johnson in Holmes *Hb.*
Tobacco virus 4. J. Johnson 1927 (in part) fide Valleau 1940.
Tobacco spot necrosis virus. J. Johnson 1925, 1927.
Solanum virus 2. Smith *Tb.*
Potato virus Yr. Köhler 1937 fide Köhler.
Marmor cucumeris var. *upsilon* Holmes *Hb.*
Cucumber-mosaic virus, potato-veinbanding strain. Holmes *Hb.*
Murialba venataenia Valleau 1940.
Potato severe mosaic virus. Samuel 1943.

Strains or suspected strains.
Potato Di Vernon streak virus. Salaman 1930 fide Bawden 1943.
Potato virus C. Bawden 1936.
Solanum virus 5. Smith *Tb.*
Solanum virus 2 (var.). Smith & Dennis 1940.

Potato Spindle Tuber. Smith *Tb.* 377; Holmes *Hb.* 111; Weiss *P.D.R.S. 155*, 110, 111.

Potato spindle tuber virus. Goss 1930.
Potato spindling tuber virus. Folsom 1923.
Potato marginal leaf roll. Quanjer 1923, Fernow 1923.
Potato virus 8. J. Johnson in Smith *Tb.*
Solanum virus 12. Smith *Tb.*
Acrogenus solani var. *vulgaris* Holmes *Hb.*
Potato spindle-tuber virus, typical strain. Holmes *Hb.*

Strain or suspected strain.
Potato unmottled curly dwarf virus. Schultz & Folsom 1923.
Potato virus 9. J. Johnson in Smith *Tb.*
Solanum virus 13. Smith *Tb.*
Acrogenus solani var. *severus* Holmes *Hb.*
Potato spindle-tuber virus, unmottled curly-dwarf strain. Holmes *Hb.*

Potato Top Necrosis, caused by strains of Potato viruses X, Y, and A in different potato varieties.

Potato Unmottled Curly Dwarf, caused by Potato spindle tuber virus.

Potato Veinal Mosaic. Smith *Tb.* 353; Holmes *Hb.* 47; Weiss *P.D.R.S. 155*, 93.
Mild Mosaic (Amer.).

Potato virus A. Murphy & McKay 1932.
Super-mild mosaic virus. Quanjer fide Smith *Tb.*
Potato veinal mosaic virus. Murphy 1936.
Potato virus P. Köhler 1936 fide Köhler 1937.

Solanum virus 3. Smith *Tb.*
Marmor solani Holmes *Hb.*
Potato mild-mosaic virus. Holmes *Hb.*

Potato Witches' Broom. Smith *Tb.* 385; Holmes *Hb.* 7; Weiss *P.D.R.S. 155,* 112.
Potato witches' broom virus. Hungerford & Dana 1924.
Tomato witches' broom virus. Young & Morris 1928.
Tobacco witches' broom virus. Young 1929.
Potato virus 11. J. Johnson in Smith *Tb.*
Solanum virus 15. Smith *Tb.*
Chlorogenus solani Holmes *Hb.*
Chlorophthora solani (Holmes) McKinney 1944.

Potato Yellow Dwarf. Smith *Tb.* 388; Holmes *Hb.* 94; Weiss *P.D.R.S. 155,* 114.
Potato yellow dwarf virus. Barrus & Chupp 1922.
Potato virus 5. J. Johnson in Smith *Tb.*
Solanum virus 16. Smith *Tb.*
Marmor vastans Holmes *Hb.*
Marmor vastans var. *vulgare* Black 1940.
New York potato yellow dwarf virus. Black 1941.
Aureogenus vastans var. *vulgare* Black 1944.
Strains or suspected strains.
Marmor vastans var. *lethale* Black 1940.
Aureogenus vastans var. *lethale* Black 1944.
New Jersey potato yellow dwarf virus. Black 1944.
Marmor vastans var. *agalliae* Black 1941.
Aureogenus vastans var. *agalliae* Black 1944.

Primula Mosaic. Brierley *P.D.R.S. 150,* 448.
Primula mosaic virus.
Primula obconica mosaic virus. Tompkins & Middleton 1941.

Privet Variegation. Holmes *Hb.* 52; Brierley *P.D.R.S. 150,* 448.
Privet variegation virus. Brierley 1944.
Ligustrum infectious chlorosis [virus]. Baur 1907.
Marmor ligustri Holmes *Hb.*
Ligustrum-mosaic virus. Holmes *Hb.*

Prunus Vein-clearing, see Cherry Vein-clearing.

Rape Savoy. Holmes *Hb.* 133.
Rape savoy virus. Holmes *Hb.*
Swede mosaic virus. Pape 1935 fide Holmes *Hb.*
? Rape mosaic virus. Kaufmann 1936.
Savoia napi Holmes *Hb.*

Raspberry Decline. Zeller & Braun, *Phytopathology,* xxxiii, 156–161, 1943.
Raspberry decline virus.
Raspberry decline disease virus. Zeller & Braun 1943.
Minuor ruborum Zeller & Braun 1943.
Rubus virus 8. Zeller & Braun 1943.

RASPBERRY LEAF CURL.[1]

ALPHA. Smith *Tb.* 121; Holmes *Hb.* 121.

Raspberry alpha leaf-curl virus.

Raspberry leaf-curl [virus]. Green 1895.
Raspberry curl virus. Melchers 1914.
Raspberry yellows virus. Melchers 1914.
Raspberry alpha-curl virus. Bennett 1930.
Raspberry virus 1A. J. Johnson in Smith *Tb.*
Rubus virus 3. Smith *Tb.*
Corium rubi var. *alpha* Holmes *Hb.*
Raspberry leaf-curl virus, alpha strain. Holmes *Hb.*

BETA. Smith *Tb.* 121; Holmes *Hb.* 122.

Raspberry beta leaf-curl virus.

Raspberry beta-curl virus. Bennett 1930.
Raspberry virus 1B. J. Johnson in Smith *Tb.*
Rubus virus 3A. Smith *Tb.*
Corium rubi var. *beta* Holmes *Hb.*
Raspberry leaf-curl virus, beta strain. Holmes *Hb.*

RASPBERRY MOSAIC. Smith *Tb.* 112, 115; Holmes *Hb.* 80.

[On *Rubus strigosus* and *R. occidentalis* (American red and black raspberries and 'purple' hybrids. The disease (Raspberry mosaic 1, Raspberry Mild Mosaic 2, Raspberry Severe Mosaic 2, Harris 1940) on *R. idaeus* (European red raspberry) is distinct, fide Harris.]

Raspberry mosaic virus. Dodge & Wilcox 1926.

Red raspberry mosaic virus Rankin & Hockey 1922.
Raspberry virus 1 (green mottle mosaic or green mosaic). Cooley 1936.
Red raspberry severe mosaic virus. Bennett 1932.
Raspberry virus 2. J. Johnson in Smith *Tb.*
Rubus virus 1. Smith *Tb.*
Marmor rubi Holmes *Hb.*
Poecili rubi (Holmes) McKinney 1944.

Strains or suspected strains.

Red raspberry mild mosaic virus. Bennett 1927, 1932.
Raspberry mosaic virus 3. Rankin 1931.
Red raspberry medium severe mosaic virus. Bennett 1932.
Raspberry yellow mosaic virus. Bennett 1927.
Raspberry mosaic virus 2. Rankin 1931.
Raspberry virus 2. Cooley 1936.
Raspberry virus 2B. J. Johnson in Smith *Tb.*
Rubus virus 2.

RASPBERRY NECROTIC FERN LEAF MOSAIC. Chamberlain (G. C.), *Sci. Agric.*, xxii, 119–124, 1941.

Raspberry necrotic fern leaf mosaic virus. Chamberlain 1941.

RASPBERRY STREAK. Smith *Tb.* 123; Holmes *Hb.* 124.

Raspberry streak virus. Zeller 1923.

Raspberry eastern blue stem virus. Wilcox 1922.
Raspberry rosette virus. Rankin 1923.
Raspberry severe streak virus. Rankin 1931.

[1] Raspberry (*Rubus idaeus*) leaf-curl of R. V. Harris is distinct from the analogous American diseases Raspberry alpha and beta curl.

Raspberry leaf-curl virus 3. Rankin 1931.
Raspberry virus 3. J. Johnson in Smith *Tb.*
Rubus virus 4. Smith *Tb.*
Nanus orientalis Holmes *Hb.*

Strain or suspected strain.
Raspberry mild streak virus. Rankin 1931.
Raspberry leaf-curl virus 4. Rankin 1931.

Raspberry Yellow Blotch-Curl. Chamberlain (G. C.), *Canad. J. Res.*, Sect. C, xvi, 118–124, 1928.
Raspberry yellow blotch-curl virus. Chamberlain 1938.

Rice Dwarf. Smith *Tb.* 450; Holmes *Hb.* 64.
Rice dwarf virus.
Rice stunt disease virus. Takata 1896.
Rice dwarf disease virus. Fukushi 1931.
Rice mosaic virus. fide Holmes *Hb.*
Oryza virus 1. Smith *Tb.*
Marmor oryzae Holmes *Hb.*

Rice Stripe. Smith *Tb.* 454.
Rice stripe virus.
Rice stripe disease virus. Kurubayashi 1931.
Oryza virus 1. Smith *Tb.*

Robinia Brooming. Smith *Tb.* 184; Holmes *Hb.* 13.
Robinia brooming virus.
Locust witches' broom [virus]. Waters 1898, Holmes *Hb.*
Black locust brooming disease [virus]. Hartley & Haasis 1929.
Robinia virus 1. Smith *Tb.*
Chlorogenus robiniae Holmes *Hb.*
Polycladus robiniae (Holmes) McKinney 1944.

Rose Mosaic. Smith *Tb.* 152; Holmes *Hb.* 74; Brierley *P.D.R.S. 150*, 470; *ibid. 158*, 190.
Rose mosaic virus. Weiss & McWhorter 1928.
Rose mosaic-like mottling [virus]. Norton 1909.
Rose infectious chlorosis virus. White 1928.
Rosa virus 1. Smith *Tb.*
Marmor rosae Holmes *Hb.*

Rose Streak. Smith *Tb.* 155; Holmes *Hb.* 75; Brierley *P.D.R.S. 150*, 171; *ibid. 158*, 190.
Rose streak virus. Brierley 1935.
Rosa virus 4. Smith *Tb.*
Marmor veneniferum Holmes *Hb.*

Rose Wilt. Smith *Tb.* 154; Holmes *Hb.* 73; Brierley *P.D.R.S. 150*, 472; *ibid. 158*, 190.
Rose wilt virus. Grieve 1931.
Rose die-back virus. Grieve 1931.
Rosa virus 3. Smith *Tb.*
Marmor flaccumfaciens Holmes *Hb.*

Rose Yellow Mosaic. Smith *Tb.* 154.
Rose yellow mosaic virus. Brierley 1935.
Rosa virus 2. Smith *Tb.*

Sandal Leaf-curl Mosaic. Smith *Tb.* 201; Holmes *Hb.* 94; Brierley *P.D.R.S. 158*, 190.
Sandal leaf-curl mosaic virus. Venkata Rao 1932.
Santalum virus 2. Smith *Tb.*
Marmor santali Holmes *Hb.*

Sandal Spike. Smith *Tb.* 196; Holmes *Hb.* 8; Brierley *P.D.R.S. 158*, 191.
Sandal spike virus.
Sandal spike disease virus.
Sandal spike rosette virus. Venkata Rao & Iyengar 1934.
Santalum virus 1. Smith *Tb.*
Chlorogenus santali Holmes *Hb.*
Sandal pendulous spike virus. Venkata Rao & Iyengar 1934 fide Brierley 1944.
Santalum virus 1A. Smith *Tb.*

Soy-Bean Mosaic. Smith *Tb.* 164; Weiss *P.D.R.S. 154*, 60.
Soy-bean mosaic virus. Gardner & Kendrick 1921.
Soybean virus 1. Pierce 1935.
Soja virus 1. Smith *Tb.*

Spinach Mosaic, caused by Cucumber mosaic virus.

Stock Mosaic. Smith *Tb.*; Holmes *Hb.* 71; Brierley *P.D.R.S. 150*, 451, 453.
Stock mosaic virus.
Stock-breaking virus. Tompkins 1934.
Stock mosaic (mild and severe) viruses. Tompkins 1939.
Matthiola virus. Smith *Tb.*
Marmor matthiolae Holmes *Hb.*

Strawberry Crinkle. Smith *Tb.* 105; Holmes *Hb.* 78.
Strawberry crinkle virus. Zeller & Vaughan 1932.
Strawberry severe crinkle virus. Harris 1937.
Strawberry virus 4. J. Johnson in Smith *Tb.*
Fragaria virus 2. Smith *Tb.*
Marmor fragariae Holmes *Hb.*

Strawberry Dwarf. Smith *Tb.* 100.
Strawberry dwarf virus. Plakidas 1928.
Strawberry virus 3. J. Johnson in Smith *Tb.*
Fragaria virus 4. Smith *Tb.*

Strawberry Stunt. Zeller & Weaver, *Phytopathology*, xxxi, 849–851, 1941.
Strawberry stunt virus. Zeller & Weaver 1941.
Fragaria virus 5. Zeller & Weaver 1941.
Nanus cupuliformans Zeller & Weaver 1941.

Strawberry Witches' Broom. Smith *Tb.* 108; Holmes *Hb.* 128.
Strawberry witches' broom virus. Zeller 1927.
Strawberry virus 2. J. Johnson in Smith *Tb.*
Fragaria virus 3. Smith *Tb.*
Nanus fragaria Holmes *Hb.*
Blastogenus fragariae (Holmes) McKinney 1944.

Strawberry Yellow Edge. Smith *Tb.* 101; Holmes *Hb.* 79.
Strawberry yellow edge virus. Harris 1933.
Strawberry yellows virus. Plakidas 1926 fide Harris.
Strawberry xanthosis virus. Plakidas 1927.
Strawberry virus 1. J. Johnson in Smith *Tb.*
Fragaria virus 1. Smith *Tb.*
Marmor marginans Holmes *Hb.*

Sugar Beet, see Beet.

Sugar-cane Dwarf. Smith *Tb.* 437; Holmes *Hb.* 109.
Sugar-cane dwarf virus.
Sugar-cane dwarf disease virus. Bell 1932, Storey 1936.
Saccharum virus 5. Smith *Tb.*
Galla queenslandiensis Holmes *Hb.*

Sugar-cane Fiji Disease. Smith *Tb.* 432; Holmes *Hb.* 106.
Sugar-cane Fiji disease virus. [Muir 1910], Kunkel 1928, Ocfemia 1932, Montgomery & Bell 1933.
Saccharum virus 2. Smith *Tb.*
Galla fijiensis Holmes *Hb.*

Sugar-cane Mosaic. Smith *Tb.* 424; Holmes *Hb.* 60.
Sugar-cane mosaic virus. Brandes 1919.
Sugar-cane yellow stripe disease [virus]. Wakker & Went 1898 fide Smith *Tb.*
Sugar-cane mottling disease [virus]. Stevenson 1917 fide Smith *Tb.*
Corn mosaic virus. Brandes 1920.
Grass mosaic virus. Brandes & Klaphaak 1923, Kunkel 1928.
Sugar-cane virus 1. J. Johnson in Smith *Tb.*
Saccharum virus 1. Smith *Tb.*
Marmor sacchari Holmes *Hb.*
Strains or suspected strains.
Maize and sorghum mosaic virus. Storey 1929.
Transvaal grass mosaic virus. Storey 1929.
Saccharum virus 1A. Smith *Tb.*
Sugar-cane mosaic type 1, 2, 3, 4 viruses. Summers 1934.
Saccharum virus 1B, 1C, 1D, 1E. Smith *Tb.*
Sugar-cane severe mosaic virus. Tims 1935.
Saccharum virus 1F. Smith *Tb.*
'Agaul' sugar-cane mosaic virus. Storey 1936.
Saccharum virus 1G. Smith *Tb.*

Sugar-cane Sereh Disease. Smith *Tb.* 436; Holmes *Hb.* 129.
Sugar-cane sereh disease virus. [Wakker & Went 1898, Kunkel 1928].
Saccharum virus 3. Smith *Tb.*
Nanus sacchari Holmes *Hb.*

Sugar-cane Streak. Smith *Tb.* 437.
Sugar-cane streak virus.
Sugar-cane 'R.P.8' streak virus. Shepherd 1929, Storey 1936.
Saccharum virus 4. Smith *Tb.*

Tea Phloem Necrosis. Bond, *Ann. appl. Biol.*, xxxi, 40–47, 1944; Brierley *P.D.R.S. 158*, 171.
Tea phloem necrosis virus. Bond 1944.
Camellia virus 1. Bond 1944.

Tobacco Broad Ring Spot. J. Johnson & Fulton, *Phytopathology*, xxxii, 605–612, 1942.

Tobacco broad ring spot virus. J. Johnson & Fulton 1940.

Tobacco Coarse Etch. E. M. Johnson, *Bull. Ky agric. Exp. Sta.* 306, p. 300, 1930.

Tobacco coarse etch virus. E. M. Johnson 1930.

Coarse-etched (virus). Valleau & E. M. Johnson 1928.

Tobacco Etch. Smith *Tb.* 259; Holmes *Hb.* 40; Bawden & Kassanis, *Ann. appl. Biol.*, xxviii, 107, 1941.

Tobacco etch virus. E. M. Johnson 1930.

Datura Z-mosaic virus. Blakeslee 1921 fide Holmes *Hb.*

Tomato etch virus. fide Valleau 1940.

Tobacco virus 13. J. Johnson in Smith *Tb.*

Nicotiana virus 7. Smith *Tb.*

Marmor erodens var. *vulgare* Holmes *Hb.*

Tobacco-etch virus, typical strain. Holmes *Hb.*

Foliopellis erodens (Holmes) Valleau 1940.

Strains or suspected strains.

Tobacco etch (Georgia). E. M. Johnson 1930.

Tobacco etch+virus. E. M. Johnson 1930.

Tobacco mild etch virus. Bawden & Kassanis 1941.

Tobacco severe etch virus. E. M. Johnson 1930, Bawden & Kassanis 1941.

Tobacco virus 13C. J. Johnson in Holmes *Hb.*

Marmor erodens var. *severum* Holmes *Hb.*

Tobacco-etch virus, severe-etch strain. Holmes *Hb.*

Tobacco Green Mosaic. Smith *Tb.* 258; Thornberry & McKinney 1939; McKinney 1944.

Tobacco green mosaic virus. McKinney 1929.

Tobacco virus 12. J. Johnson in Smith *Tb.*

Nicotiana virus 6. Smith *Tb.*

Tobacco mild-dark-green mosaic virus. Thornberry & McKinney 1939; McKinney 1944.

Marmor constans McKinney 1944.

Tobacco Leaf Curl. Smith *Tb.* 262; Holmes *Hb.* 115.

Tobacco leaf-curl virus. Storey 1932.

Tobacco Faltenzwerg [virus]. Peters & Schwarz 1912 fide Thung 1932.

Tobacco krekoh [virus]. Keuchenius 1915 fide Thung 1932.

Tobacco gilah [virus]. Jochems 1926 fide Thung 1932.

Tobacco crinkle [virus]. Roberts 1930 fide Thung 1932.

Tobacco crinkly dwarf. Storey 1931, Moore 1933 fide Thung 1932.

? Tobacco cabbaging virus. Storey 1932.

Tobacco curly leaf virus. Storey 1932.

Tobacco 'frenching' virus. Storey 1932.

Tobacco (gewone) kroepoek virus. [Keuchenius 1915] Thung 1932.

Tobacco kroesblaar virus. Moore 1933.

Tobacco leaf-curl virus 1. Pal 1937.

Tobacco virus 16. J. Johnson in Smith *Tb.*

Nicotiana virus 10. Smith *Tb.*

Ruga tabaci Holmes *Hb.*

Strains or suspected strains.

Tobacco leaf curl viruses 2, 3 (= tobacco heldere kroepoek virus. Thung 1932), and 4. Pal 1937.

Tobacco krulziekte (leaf curl) virus. Thung 1932.

Tobacco Mosaic. Smith *Tb.* 230; Holmes *Hb.* 17.

Tobacco mosaic virus. Clinton 1909, Allard 1914.

Mosaikkrankheit des Tabaks [virus]. Mayer 1886.

Pockenkrankheit des Tabaks [virus]. Iwanowski 1890.

Tobacco calico virus. Clinton 1903.

Tobacco mosaic virus A. Fernow 1926.

Tobacco ordinary mosaic virus. J. Johnson 1926.

Tobacco virus 1. Johnson 1927.

Tobacco true mosaic virus. Valleau & E. M. Johnson 1927.

Tobacco 'french' (or 'dry weather french') virus. Valleau & E. M. Johnson 1927.

Tobacco severe mosaic type 1 virus. E. M. Johnson 1930.

Pepper mosaic virus. E. M. Johnson 1930.

Typical tobacco mosaic virus. Duggar 1933.

Ordinary or mild tomato mosaic virus. Ainsworth 1933.

Tobacco distorting mosaic virus. Duggar & B. Johnson 1933, Holmes 1934.

Ordinary field type tobacco mosaic virus. Kunkel 1934.

Tobacco virus 1A. Thung 1935.

Nicotiana virus 1. Smith *Tb.*

Tobacco mosaic virus, green-mottling distorting-type strain. Jensen 1937 fide Jensen.

Marmor tabaci var. *vulgare* Holmes *Hb.*

Tobacco mosaic virus, typical strain. Holmes *Hb.*

Protobios mosaicus var. *tabaci* d'Herelle.

Musivum tabaci (Holmes) Valleau 1940.

Marmor tabaci (Holmes ex Valleau) McKinney 1944.

Strains or suspected strains.

Plantago mosaic virus.

Marmor tabaci var. *plantaginis* Holmes 1941.

Tobacco mosaic virus rib-grass strain. Holmes 1941.

Plantago virus. Valleau & Johnson 1943.

Tobacco attenuated mosaic virus. J. Johnson 1926.

Tobacco medium mosaic virus. J. Johnson 1927.

Tobacco virus 7. J. Johnson.

Tobacco concentrated yellow mosaic virus type 1 and 2. McKinney 1931.

Tobacco mild mosaic virus types A and B. Matsumoto & Tateoka 1940.

Tobacco mild mosaic virus types 1 and 2. E. M. Johnson 1930.

Tobacco mild mosaic, white mosaic (= virus VIA, Thung 1938), distorting mosaic 1, ringspot necrosis, yellow bleaching tobacco ring mosaic, and severe mosaic viruses. Thung 1935.

Tobacco mosaic viruses VIb and VIc. Thung 1938.

Tobacco-mosaic virus, localized yellow-lesion strain. Holmes *Hb.*

Marmor tabaci var. *immobile* Holmes *Hb.*

Tobacco-mosaic virus, masked-symptom strain. Holmes 1934.

Nicotiana virus 1D. Smith *Tb.*

Marmor tabaci var. *obscurum* Holmes *Hb.*

Tobacco-mosaic virus, minute yellow-lesion strain. Holmes *Hb.*

Tobacco mosaic virus, strain 104. Jensen 1937.

Marmor tabaci var. *artum* Holmes *Hb.*

Tobacco-mosaic virus, mottling strain. Holmes 1934.
Tobacco mosaic virus, non-distorting, green-mottling strain (virus No. 502). Jensen 1937 fide Jensen.
Tobacco-mosaic virus necrotic-lesion strain. Holmes *Hb.*
Tobacco mosaic virus, strain J 14. Jensen 1937.
Marmor tabaci var. *lethale* Holmes *Hb.*
Tobacco mosaic virus, strains 101, 302, 111, 9, 3, 14. Jensen 1937.
Tobacco non-burning virus. Valleau & E. M. Johnson 1935.
Tobacco ring mosaic virus. E. M. Johnson 1930.
Tomato ring mosaic streak virus. Ainsworth *et al.* 1934.
Lycopersicum virus 2. Smith *Tb.*
Tobacco Rotterdam B disease virus. Jochems 1933 fide Thung 1938.
Tobacco virus 17. J. Johnson in Smith *Tb.*
Nicotiana virus 9. Smith *Tb.*
Tobacco speckled mosaic virus. J. Johnson 1926.
Tobacco virus 2. J. Johnson 1927.
Nicotiana virus 2. Smith *Tb.*
Tobacco severe mosaic type 2 virus. E. M. Johnson 1930.
Tobacco veinbanding necrosis virus. Chamberlain 1940.
Tobacco white mosaic virus. E. M. Johnson 1930.
Tobacco yellow mosaic virus. McKinney 1943.
Tobacco yellow mosaic virus, isolations No. 3, No. 107. Jensen 1930.
Tobacco yellow mosaic mutant virus BSY. McKinney 1943.
Tomato aucuba mosaic virus. Bewley 1923, J. H. Smith 1928.
Tobacco yellow mosaic virus. McKinney 1926, ? E. M. Johnson 1930.
Tobacco virus 6. J. Johnson 1937 fide Ainsworth *et al.* 1934.
Tobacco aucuba mosaic virus. Kunkel 1932.
Tobacco yellow mosaic virus, isolation No. 102. Jensen 1933.
Tomato yellow mosaic virus. Ainsworth *et al.* 1934.
Tomato aucuba mosaic virus AY. Caldwell 1935.
Tobacco mosaic virus, yellow-mottling aucuba-type strain (virus No. 108). Jensen 1937 fide Jensen.
Nicotiana virus 1C. Smith *Tb.*
? *Nicotiana* virus 1B. Smith *Tb.*
Tomato yellow leaf virus. Smith *Tb.*
Marmor tabaci var. *aucuba* Holmes *Hb.*
Tobacco mosaic virus, tomato aucuba mosaic strain. Holmes *Hb.*
Tomato aucuba mosaic virus AG. Caldwell 1935.
Tobacco mosaic virus, green-mottling aucuba-type strain (virus No. 501). Jensen 1937 fide Jensen 1937.
Tomato aucuba mosaic virus, attenuated strains. Kunkel 1934.
Tomato enation mosaic virus. Ainsworth 1937.
Nicotiana virus 1A. Smith *Tb.*
Marmor tabaci var. *deformans* Holmes *Hb.*
Tobacco-mosaic virus, enation-mosaic strain. Holmes *Hb.*
Tomato fruit stripe virus. L. K. Jones 1940.
Tomato leaf-shrivelling virus. Doolittle & Beecher 1942.
Marmor tabaci var. *siccans* Doolittle & Beecher 1942.
Tomato stem necrosis virus. J. Johnson 1927.
Tobacco virus 9. J. Johnson 1927.
Tomato streak virus.
Tomato stripe, streak, or glasshouse streak virus. Jarrett 1930.
Tomato single-virus streak virus. Ainsworth *et al.* 1934.

Tomato streak virus No. 1. Ainsworth *et al.* 1934.
Tomato virus 4. J. Johnson in Smith *Tb.*
Lycopersicum virus 1. Smith *Tb.*
Marmor tabaci var. *canadense* Holmes *Hb.*
Tobacco-mosaic virus, single-virus streak strain. Holmes *Hb.*
Severe mottle streak virus. Selman 1941.
Mild-mottle streak virus. Selman 1941.
Tomato streak virus, yellow & green strains. Smith 1935.
Tomato streak virus 1, Ontario strain. Berkeley 1936.

Tobacco Necrosis. Smith *Tb.* 265; Holmes *Hb.* 86. [Includes a number of serologically unrelated viruses, see Bawden, *Brit. J. exp. Path.*, xxii, 59–70, 1941.]
Tobacco necrosis virus. Smith & Bald 1935; Price 1938.
Nicotiana virus 11. Smith *Tb.*
Marmor lethale Holmes *Hb.*

Tobacco Ring Spot. Smith *Tb.* 269; Holmes *Hb.* 98.
Tobacco ring spot virus. Fromme *et al.* 1927.
Tobacco hieroglyphics [virus]. 1923.
Tobacco ring spot No. 1 virus. Price 1936.
Tobacco virus 10. J. Johnson in Smith *Tb.*
Nicotiana virus 12. Smith *Tb.*
Annulus tabaci var. *virginiensis* Holmes *Hb.*
Tobacco-ringspot virus, typical strain. Holmes *Hb.*
Marmor annularium McKinney 1944.
Strains or suspected strains.
Bergerac ringspot virus. Smith 1935.
Nicotiana virus 14. Smith *Tb.*
Annulus tabaci var. *kentuckiensis* Holmes *Hb.*
Tobacco ringspot virus, green-ringspot strain. Holmes *Hb.*
Tobacco ringspot No. 2 virus. Price 1936.
Nicotiana virus 13. Smith *Tb.*
Annulus zonatus Holmes *Hb.*
Tomato-ringspot virus. Holmes *Hb.*
Yellow ringspot virus. Valleau 1932, Price 1936.
Nicotiana virus 12A. Smith *Tb.*
Annulus tabaci var. *auratus* Holmes *Hb.*
Tobacco ringspot virus, yellow-ringspot strain. Holmes *Hb.*

Tobacco Streak. Smith *Tb.* 259; Holmes *Hb.* 103.
Tobacco streak virus. J. Johnson 1936.
? Tobacco vein-streak virus. Jochems 1933.
Tobacco viruses B5 and B6. Thung 1933.
Tobacco virus 18. J. Johnson in Smith *Tb.*
Nicotiana virus 8. Smith *Tb.*
Annulus orae Holmes *Hb.*
Tractus orae (Holmes) Valleau 1940.
Strain or suspected strain.
Tobacco Streifen- and Kräuselkrankheit virus. Böning 1928.
Tobacco virus 11. J. Johnson in Smith *Tb.*
Nicotiana virus 5. Smith *Tb.*

Tobacco Yellow Dwarf. Hill, *J. Coun. sci. industr. Res. Aust.*, x, 228–230, 1937; *ibid.* 1941, 1942.
Tobacco yellow leaf dwarf virus. Hill 1937.

Tomato Aucuba Mosaic, caused by Tobacco mosaic virus (str.).

Tomato Big Bud. Smith *Tb.* 322; Holmes *Hb.* 107.
Tomato big bud virus. Samuel *et al.* 1933.
Tomato rosette virus. [Cobb 1902] Samuel *et al.* 1933.
Tomato blue top virus. Samuel *et al.* 1933.
Tomato bunchy top virus. Samuel *et al.* 1933.
Tomato fruit woodiness virus. Ryjkoff *et. al.* 1933 fide Ryjkoff 1934.
Virescence virus. Ryjkoff 1934, Hill 1943.
Tomato stolbur virus. Michailowa 1935.
Tobacco big bud virus. Hill 1937.
Lycopersicum virus 5. Smith *Tb.*
Galla australiensis Holmes *Hb.*

Tomato Bunchy Top. Smith *Tb.* 322.
Tomato bunchy top virus. McClean 1931, 1935.
Tomato virus 2. J. Johnson in Smith *Tb.*
Lycopersicum virus 6. Smith *Tb.*

Tomato Bushy Stunt. Smith *Tb.* 315; Holmes *Hb.* 30.
Tomato bushy stunt virus. Ainsworth 1936.
Lycopersicum virus 4. Smith *Tb.*
Marmor dodecahedron Holmes *Hb.*

Tomato Enation Mosaic, caused by Tobacco mosaic virus (str.).

Tomato Fern Leaf, caused by Cucumber mosaic virus.

Tomato Mosaic, caused by Tobacco mosaic virus.

Tomato Spotted Wilt. Smith *Tb.* 296; Holmes *Hb.* 136.
Tomato spotted wilt virus. Samuel *et al.* 1930.
'T.S.W.' virus. (Pethybridge in) Ainsworth *et al.* 1934.
Tomato virus. Azevedo 1936, fide Costa & Forster 1941.
Tomato virus 1. J. Johnson in Smith *Tb.*
Lycopersicum virus 3. Smith *Tb.*
Lethum australiense var. *typicum* Holmes *Hb.*
Tomato spotted-wilt virus, typical strain. Holmes *Hb.*
Pineapple yellow spot virus. Illingworth 1931, Linford 1932, fide Sakimura, 1940.
Pineapple side rot [virus]. Sideris 1927.
Ananas virus 1. Smith *Tb.*
Strains or suspected strains.
Kromnek virus. Moore 1933.
Tobacco wilt [virus]. Lounsbury 1906.
Kat River disease or wilt virus. Moore 1933.
Tobacco corcova virus. [Fawcett 1921] Soriano 1931, Fawcett 1940.
Tomato corcova virus. Fawcett 1933, 1940.
Tomato black pest virus. Fawcett 1940.
? Potato vira-cabeça virus. Costa & Kiehl 1938, Fawcett 1940.
Tomato tip-blight virus.
Oregon tip-blight of tomato virus. McWhorter & Milbrath 1935, 1938.

Tomato die-back streak virus. Shapovalov 1934.
Lethum australiense var. *lethale* Holmes *Hb.*
Tomato spotted-wilt virus, tip-blight strain. Holmes *Hb.*

Tomato Streak, caused by Tobacco mosaic virus (str.).

Tomato Streak, Mixed-virus, caused by Tobacco mosaic virus+Potato virus X.

Tulip Breaking. Brierley *P.D.R.S. 150*, 476.
Tulip breaking virus.
Tulipa virus 1. Smith em. Brierley & F. F. Smith 1944.
Marmor tulipae Holmes em. Brierley & F. F. Smith 1944.
Lily mottle viruses. Brierley & F. F. Smith 1944.
[For a longer list of synonyms and possible synonyms see Brierley (*loc. cit.*).]

Turnip Mosaic. Smith *Tb.* 12; Holmes *Hb.* 70.
Turnip mosaic virus. Gardner & Kendrick 1921, Schultz 1921, Chamberlain 1936, Tompkins 1938.
Mustard mosaic virus. Schultz 1921.
Chinese cabbage mosaic virus. Schultz 1921.
Turnip virus 1. Hoggan & J. Johnson in Smith *Tb.*
Crucifer mosaic virus. Hoggan & J. Johnson 1935.
Brassica virus 2. Smith *Tb.*
Marmor brassicae Holmes *Hb.*
Crucifer mosaic virus. Clayton 1930 fide Prentice *in litt.*
Brassica virus 4. Smith *Tb.*
Chinese cabbage mosaic virus. Tompkins & Thomas 1938 fide Holmes *Hb.*
Turnip mosaic virus. Tompkins 1938 fide Holmes *Hb.*
Radish mosaic virus. Tompkins 1939 fide Prentice *in litt.*
Strains or suspected strains.
Cabbage virus A. Walker, LeBeau, & Pound 1945.
? Cabbage black ring spot virus (q.v.).

Vine Mosaic. Smith *Tb.* 202; Holmes *Hb.* 61.
Vine mosaic virus. Stranak 1931.
Vitis virus 1. Smith *Tb.*
Marmor viticola Holmes *Hb.*

Wallflower Breaking, caused by Cabbage black ringspot virus.

Wheat Mosaic. Smith *Tb.* 448; Holmes *Hb.* 61; McKinney, *J. Wash. Acad. Sci.*, xxiv, 323–325, 1944.
Wheat mosaic virus. McKinney 1925.
Wheat rosette virus. McKinney 1923.
Wheat virus 1. McKinney 1937.
Wheat green mosaic (or mosaic rosette) virus. McKinney 1937.
Triticum virus 1. Smith *Tb.*
Marmor tritici Holmes *Hb.*
Marmor tritici var. *typicum* McKinney 1944.
Wheat mosaic-rosette virus. McKinney 1944.
Strains or suspected strains.
Wheat virus 2 (green mosaic). McKinney 1937.
Wheat virus 3. McKinney 1931, 1937.
Triticum virus 1A. Smith *Tb.*
Marmor tritici var. *fulvum* McKinney 1944.
Prairie wheat yellow-mosaic virus. McKinney 1944.

Wheat virus 4 (light green mosaic). McKinney 1937.
Wheat green mosaic virus. Wada & Fukano 1937.
Marmor campestre var. *typicum* McKinney 1944.
Plains' wheat green-mosaic virus. McKinney 1944.
Wheat virus 5 (yellow mosaic). McKinney 1937.
Marmor campestre var. *galbanum* McKinney 1944.
Plains' wheat yellow-mosaic virus. McKinney 1944.
Wheat virus 6 (mild streak mosaic). McKinney 1937.
Marmor virgatum var. *viride* McKinney 1944.
Wheat green streak-mosaic virus. McKinney 1944.
Wheat virus 7 (yellow streak mosaic). McKinney 1937.
Marmor virgatum var. *typicum* McKinney 1944.
Wheat yellow streak-mosaic virus. McKinney 1944.

Wheat Mosaic, Winter. McKinney, *J. Wash. Acad. Sci.*, xxxiv, 327, 1944.
Winter wheat mosaic virus. Zazhurilo & Sitnikova 1939.
Russian wheat-mosaic virus. McKinney 1944.
Fractilinea tritici McKinney 1944.

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'Agaul' sugar-cane mosaic virus, see Sugar-cane mosaic virus (str.).
Agropyron green mosaic virus, see *Agropyron* mosaic virus (str.).
Agropyron mosaic virus, 515.
Agropyron yellow mosaic virus, see *Agropyron* mosaic virus (str.).
Alfalfa dwarf virus, see Lucerne dwarf virus.
Alfalfa mosaic virus, see Lucerne mosaic virus.
Alfalfa mosaic virus 1, see Lucerne mosaic virus.
Alfalfa mosaic virus 1A, see Lucerne mosaic virus (str.).
Alfalfa mosaic virus 1B, see Lucerne mosaic virus (str.).
Alfalfa ringspot virus, see Lucerne mosaic virus.
Alfalfa virus 1, see Lucerne mosaic virus.
Alfalfa virus 2, see Lucerne mosaic virus.
Alfalfa witches' broom (Amer.), see Lucerne witches' broom.
Allium virus 1, see Onion yellow dwarf virus.
Alsike clover mosaic virus, 520.
Alsike clover mosaic virus 1, see Alsike clover mosaic virus.
Alsike clover mosaic virus 2, see Alsike clover mosaic virus (str.).
Alsike clover virus 1, see Alsike clover mosaic virus.
Alsike virus 1, see Alsike clover mosaic virus.
American mild mosaic, see Potato crinkle.
Ananas virus 1, see Tomato spotted wilt virus.
Anemone alloiophylly virus, 515.
Anemone virus 1, see Anemone alloiophylly virus.
Annulus orae, see Tobacco streak virus.
Annulus tabaci var. *auratus,* see Tobacco ring spot virus (str.).
Annulus tabaci var. *kentuckiensis,* see Tobacco ring spot virus (str.).
Annulus tabaci var. *virginiensis,* see Tobacco ring spot virus.
Annulus zonatus, see Tobacco ring spot virus (str.).
Apium virus 1, see Celery mosaic virus.
Apple infectious variegation virus, see Apple mosaic virus.
Apple mosaic virus, 515.
Arachis virus 1, see Groundnut rosette virus.
Aster yellows virus, 515.
Aster yellows virus, celery yellows strain, see Aster yellows virus (str.).
Aster yellows virus, heat attenuated strain, see Aster yellows virus (str.).
Aster yellows virus, typical strain, see Aster yellows virus.
Attenuated sugar beet curly top virus, see Beet curly top virus (str.).
Aureogenus clavifolium, see Clover club leaf virus.
Aureogenus magginerva, see Clover big vein virus.
Aureogenus vastans var. *agalliae,* see Potato yellow dwarf virus (str.).
Aureogenus vastans var. *lethale,* see Potato yellow dwarf virus (str.).
Aureogenus vastans var. *vulgare,* see Potato yellow dwarf virus.

Banana bunchy top virus, 516.
Banana heart rot and infectious chlorosis virus, see Cucumber mosaic virus (str.).
Banana mosaic virus, see Cucumber mosaic virus (str.).
Banana virus 1, see Banana bunchy top virus.
Banana virus 2, see Cucumber mosaic virus (str.).
Bean dwarf mosaic virus, see Bean yellow mosaic virus.
Bean leaf wilt virus, 516.
Bean mosaic virus, 516.
Bean mosaic virus 4, see Southern bean mosaic virus.
Bean mosaic virus 4A, see Southern bean mosaic virus (str.).
Bean mosaic virus 'new strain', see Bean mosaic virus (str.).
Bean virus 1, see Bean mosaic virus.
Bean virus 2, see Bean yellow mosaic virus.
Bean yellow mosaic virus, 517.
Bean yellow necrosis virus, 517.
Beet curly top virus, 517.
? Beet jaunisse virus, see Beet mosaic virus and Beet yellows virus.
Beet Kräuselkrankheit virus, see Beet leaf curl virus.
Beet leaf curl virus, 517.
Beet mosaic virus, 517.
Beet mosaic virus, new str., see Beet mosaic virus (str.).
Beet savoy disease virus, 518.
Beet savoy virus, see Beet Savoy disease virus.
Beet vergelingsziekte virus, see Beet yellows virus.
Beet yellows virus, 518.
Beet yellows [virus], see Beet mosaic virus.
Bergerac ringspot virus, see Tobacco ring spot virus (str.).
Beta virus 1, see Beet curly top virus.
Beta virus 2, see Beet mosaic virus.
Beta virus 3, see Beet leaf curl virus.
Beta virus 4, see Beet yellows virus.
Beta virus 5, see Beet savoy disease virus.
Bettendorf mosaic virus, see Cucumber mosaic virus (str.).
Blackberry dwarf virus, 518.
Black-currant reversion disease virus, see Currant reversion virus.
Black locust brooming disease [virus], see *Robinia* brooming virus.
Blastogenus fragariae, see Strawberry witches' broom virus.
Blind pocket psorosis virus, see Citrus psorosis virus (str.).
Bottle-gourd (*Lagenaria vulgaris*) mosaic virus, see Cucumber mosaic virus (str.).
Brassica virus 1, see Cabbage black ring spot virus.
Brassica virus 2, see Turnip mosaic virus.
Brassica virus 3, see Cauliflower mosaic virus.
Brassica virus 4, see Turnip mosaic virus.
Broad-bean local-lesion virus, see Pea wilt virus.
Broad bean mosaic virus, see Pea mottle virus.
Broccoli mosaic virus, see Cauliflower mosaic virus.
Brome mosaic virus, 518.
Bromus inermis, see Brome mosaic virus.
Bulb-plant mosaic [virus], see Ornithogalum mosaic virus.
Bunchy top virus, see Lucerne witches' broom virus.

Cabbage black ring spot virus, 518.
Cabbage black ring spot virus, see Turnip mosaic virus.
Cabbage black ring virus, see Cabbage black ring spot virus.
? Cabbage blackspot virus, see Cabbage black ring spot virus.
Cabbage mosaic virus, see Cabbage black ring spot virus.
Cabbage mosaic virus, see Cauliflower mosaic virus.
Cabbage ring necrosis virus, 518.
Cabbage virus A, see Turnip mosaic virus (str.).
Cabbage virus B, see Cauliflower mosaic virus (str.).
Cacao swollen shoot virus, 518.
Calico or mosaic of cucumber and melon virus, see Cucumber mosaic virus.
California aster yellows virus, see Aster yellows virus (str.).
Callistephus virus 1, see Aster yellows virus.
Callistephus virus 1A, see Aster yellows virus (str.).
Camellia virus 1, see Tea phloem necrosis virus.
Canada streak virus, see Potato aucuba mosaic virus.
Cantaloupe mosaic virus, see Cucumber mosaic virus (str.).
Carpophthora lacerans, see Peach X-disease virus.
Carrot, parsnip, and parsley yellows virus, see Aster yellows virus (str.).
Cassava brown streak virus, 518.
Cassava 'Kräuselkrankheit' [virus], see Cassava mosaic virus.
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Cassava stem lesion virus, see Cassava brown streak virus.
Cauliflower mosaic virus, 519.
Cauliflower virosis virus, see Cauliflower mosaic virus (str.).
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Celery mosaic virus, see Cucumber mosaic virus (str.).
Celery southern mosaic virus, see Cucumber mosaic virus (str.).
Celery virus 1, see Cucumber mosaic virus (str.).
Celery western mosaic virus, see Celery mosaic virus.
Celery yellows virus, see Aster yellows virus (str.).
Cherry banded chlorosis, 519.
Cherry mottle leaf virus, 519.
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Cherry rusty mottle virus, 519.
Cherry vein-clearing virus, 519.
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Chilli mosaic virus, see Lucerne mosaic virus.
Chinese cabbage mosaic virus, see Turnip mosaic virus.
Chlorogenus callistephi var. *attenuatus*, see Aster yellows virus (str.).
Chlorogenus callistephi var. *californicus*, see Aster yellows virus (str.).
Chlorogenus callistephi var. *vulgaris*, see Aster yellows virus.
Chlorogenus eutetticola, see Beet curly top virus.
Chlorogenus humuli, see Hop chlorotic disease virus (str.).
Chlorogenus medicaginis, see Lucerne witches' broom virus.
Chlorogenus persicae var. *micropersica*, see Peach yellows virus (str.).
Chlorogenus persicae var. *vulgare*, see Peach yellows virus.
Chlorogenus robiniae, see *Robinia* brooming virus.

Chlorogenus santali, see Sandal spike virus.
Chlorogenus solani, see Potato witches' broom virus.
Chlorogenus vaccinii, see Cranberry false blossom virus.
Chlorophthora solani, see Potato witches' broom virus.
Chlorosis rosette, green-rosette, and type No. 3 viruses, see Groundnut rosette virus (str.).
Citrivir italicum, see Citrus infectious mottling virus.
Citrivir psorosis, see Citrus psorosis virus.
Citrivir psorosis var. *alveatum*, see Citrus psorosis virus (str.).
Citrivir psorosis var. *annulatum*, see Citrus psorosis virus (str.).
Citrivir psorosis var. *concavum*, see Citrus psorosis virus (str.).
Citrivir psorosis var. *vulgare*, see Citrus psorosis virus (str.).
Citrus infectious mottling virus, 519.
Citrus psorosis virus, 520.
Citrus psorosis A virus, see Citrus psorosis virus (str.).
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Clover big vein virus, 520.
Clover club leaf virus, 520.
Clover mosaic, Alsike, 520.
Clover vein mosaic virus, 520.
Coarse-etched (virus), see Tobacco coarse etch.
Commelina mosaic virus, see Cucumber mosaic virus (str.).
Commelina mosaic virus yellow and green strains, see Cucumber mosaic virus (str.).
Common bean mosaic virus, see Bean mosaic virus.
Common pea mosaic virus, see Pea mosaic virus.
Concave gum psorosis virus, see Citrus psorosis virus (str.).
Corium rubi var. *alpha*, see Raspberry alpha leaf curl virus.
Corium rubi var. *beta*, see Raspberry beta leaf curl virus.
Corium solani, see Potato leaf roll virus.
Corn leaf stripe virus, see Maize mosaic virus.
Corn mosaic (Amer.), see Maize mosaic.
Corn mosaic virus, see Sugar-cane mosaic virus.
Corn stripe virus, see Maize mosaic virus.
Corn virus 1, see Maize mosaic virus.
Corn virus 2, see Maize streak virus.
Cotton leaf crinkle virus, see Cotton leaf curl virus.
Cotton leaf curl virus, 520.
Cowpea mosaic virus, see Cucumber mosaic virus (str.).
Cranberry false blossom virus, 520.
Crinkle mosaic, see Potato crinkle.
Crinkly-leaf psorosis virus, see Citrus psorosis virus (str.).
Crucifer mosaic virus, see Turnip mosaic virus.
Cucumber aucuba mosaic virus, see Cucumber green-mottle mosaic virus (str.).
Cucumber green-mottle mosaic virus, 522.
? Cucumber mild mosaic virus, see Cucumber mosaic virus (str.).
Cucumber mild or ordinary mosaic virus, see Cucumber green-mottle mosaic virus.
Cucumber mosaic virus, 521.
Cucumber-mosaic virus, cowpea-mottling strain, see Cucumber mosaic virus (str.).
Cucumber mosaic virus, indicator strain, see Cucumber mosaic virus (str.).
Cucumber mosaic virus, lily-mosaic strain, see Cucumber mosaic virus (str.).
Cucumber mosaic virus, Lima-bean mosaic strain, see Cucumber mosaic virus (str.).
Cucumber-mosaic virus, potato-veinbanding strain, see Potato virus Y.
Cucumber-mosaic virus, southern celery-mosaic strain, see Cucumber mosaic virus (str.).
Cucumber mosaic virus, strain 1, see Cucumber mosaic virus.
Cucumber mosaic virus, strain 3, see Cucumber mosaic virus (str.).
Cucumber mosaic virus, strain 5 (yellow cucumber mosaic virus), see Cucumber mosaic virus (str.).
Cucumber mosaic virus, strain 6, see Cucumber mosaic virus (str.).
? Cucumber mosaic virus, strain Y, see Cucumber mosaic virus (str.).
Cucumber mosaic virus type 1, see Cucumber mosaic virus.
Cucumber mosaic virus, type 2, see Cucumber mosaic virus (str.).
Cucumber mosaic virus, type 3, see Cucumber mosaic virus (str.).
Cucumber mosaic virus, typical strain, see Cucumber mosaic virus.
Cucumber virus 1, see Cucumber mosaic virus.
Cucumber virus 2, see Cucumber mosaic virus (str.).
Cucumber virus 3, see Cucumber green-mottle mosaic virus.
Cucumber virus 4, see Cucumber green-mottle mosaic virus (str.).
Cucumber wart disease [virus], see Cucumber mosaic virus.
Cucumber white-pickle mosaic virus, see Cucumber mosaic virus.
Cucumber yellow mosaic virus, see Cucumber green-mottle mosaic virus (str.).
Cucumber yellow mosaic virus, see Cucumber mosaic virus.
Cucumber yellow-mottle mosaic virus, see Cucumber mosaic virus.
Cucumis virus 1, see Cucumber mosaic virus.
Cucumis virus 1A, see Cucumber mosaic virus (str.).
Cucumis virus 1B, see Cucumber mosaic virus (str.).
Cucumis virus 1D, see Cucumber mosaic virus (str.).
Cucumis virus 2, see Cucumber green-mottle mosaic virus.
Cucumis virus 2A, see Cucumber green-mottle mosaic virus (str.).
Cucumis virus 3, see Cucumber mosaic virus (str.).
Cucumis virus 10, see Cucumber mosaic virus (str.).
Cucurbit mosaic virus, aucuba-mosaic strain, see Cucumber green-mottle mosaic virus (str.).
Cucurbit mosaic virus, green-mottling strain, see Cucumber green-mottle mosaic virus.
Cucurbit yellow mosaic virus, see Cucumber mosaic virus (str.).
Currant reversion virus, 522.
Cuscuta latent virus, see Dodder latent mosaic virus.
Cuscuta virus 1, see Dodder latent mosaic virus.
Cuscutavir secretum, see Dodder latent mosaic virus.

Dahlia leaf curl and rosette [virus], see Dahlia mosaic virus.
Dahlia mosaic virus, 523.
Dahlia oak-leaf virus, 523.
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Dahlia runting [virus], see Dahlia mosaic virus.
Dahlia stunt or dwarf [virus], see Dahlia mosaic virus.
Dahlia virus 1, see Dahlia mosaic virus.
Dahlia virus 2, see Dahlia ring spot virus.
Dahlia virus 2A, see Dahlia ring spot virus (str.).
Dahlia virus 2B, see Dahlia ring spot virus (str.).
Dahlia virus 3, see Dahlia oak-leaf virus.
Dahlia yellow ring spot virus, see Dahlia ring spot virus (str.).
Dahlia yellows [virus], see Dahlia mosaic virus.
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Datura Z-mosaic virus, see Tobacco etch virus.
Delphinium aster yellows virus, see Aster yellows virus (str.).
Delphinium ring spot virus, see Cucumber mosaic virus (str.).
Delphinium stunt disease virus, see Cucumber mosaic virus (str.).
Delphinium virus 2, see Cucumber mosaic virus (str.).
Delphinium witches' broom disease virus, see Cucumber mosaic virus (str.).
Dewberry dwarf virus, see Blackberry dwarf virus.
Dock infectious chlorosis (virus), see Dock mosaic virus.
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'Eelworm' disease [virus], see Hop nettlehead virus.
Erigeron yellows virus, see Aster yellows virus.
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Euonymus variegation virus, 523.

Ficivir caricae, see Fig mosaic virus.
Ficus virus 1, see Fig mosaic virus.
Fig mosaic virus, 523.
Flavimacula persicae, see Peach mosaic virus.
(Flowering) Cherry banded chlorosis virus, 519.
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Fractilinea avenae, see Oat pseudorosette virus.
Fractilinea maidis, see Maize streak virus.
Fractilinea tritici, see Winter wheat mosaic virus.
Fragaria virus 1, see Strawberry yellow edge virus.
Fragaria virus 2, see Strawberry crinkle virus.
Fragaria virus 3, see Strawberry witches' broom virus.
Fragaria virus 4, see Strawberry dwarf virus.
Fragaria virus 5, see Strawberry stunt virus.
Fuggles hop mosaic disease virus, see Hop chlorotic disease virus (str.).

Galla anemones, see Anemone alloiophylly virus.
Galla australiensis, see Tomato big bud virus.
Galla fijiensis, see Sugar-cane Fiji disease virus.
Galla queenslandiensis, see Sugar-cane dwarf virus.
Galla verruca, see Peach wart virus.
Galla zeae, see Maize wallaby-ear virus.
Garden pea mosaic virus, see Pea mosaic virus.
Gelbsucht der Sommerastern virus, see Aster yellows virus.
Geranium crinkle virus, see *Pelargonium* leaf curl virus.
Geranium leaf curl virus, see *Pelargonium* leaf curl virus.
Geranium mosaic virus, see *Pelargonium* leaf curl virus.
Gossypium virus 1, see Cotton leaf curl virus.
Grass mosaic virus, see Sugar-cane mosaic virus.
Gray disease virus, see Narcissus mosaic virus.
Groundnut Kräuselkrankheit [virus], see Groundnut rosette virus.
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Healthy potato virus, see Potato virus X.
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Holodiscus virus 1, see Holodiscus witches' broom virus.
Holodiscus witches' broom virus, 524.
Hop chlorotic disease virus, 524.
? Hop infectious sterility virus, see Hop nettlehead virus.
? Hop Kräuselkrankheit or curl disease [virus], see Hop nettlehead virus.
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Humulus virus 3, see Hop chlorotic disease virus.
Humulus virus 4, see Hop chlorotic disease virus (str.).
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Infectious chlorosis [virus], see Mountain ash variegation virus.
Inops consilii, see Yellow bud mosaic virus.
Interveinal mosaic virus, see Potato virus X.
Iris mosaic virus, 524.
Iris stripe virus, see Iris mosaic virus.
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Kat River disease or wilt virus, see Tomato spotted wilt virus (str.).
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Kurrajong virus, see Lucerne witches' broom virus.

Laburnum infectious chlorosis [virus], see Laburnum variegation virus.
Laburnum mosaic virus, see Laburnum variegation virus.
Laburnum variegation virus, 524.
Lactuca virus 1, see Lettuce mosaic virus.
Latent potato virus, see Potato virus X.
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Lethum australiense var. *lethale,* see Tomato spotted wilt virus (str.).
Lethum australiense var. *typicum,* see Tomato spotted wilt virus.
Lettuce and endive yellows virus, see Aster yellows virus.
Lettuce mosaic virus, 524.
Lettuce rabbit-ear virus, see Aster yellows virus.
Lettuce Rio Grande disease, see Aster yellows virus.
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Ligustrum infectious chlorosis [virus], see Privet variegation virus.
Ligustrum-mosaic virus, see Privet variegation virus.
Lilium virus 1, see Lily rosette virus.
Lily mosaic virus, see Cucumber mosaic virus (str.).
Lily mottle viruses, see Tulip breaking virus.
Lily necrotic fleck, 524.
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Lily virus, cucumber mosaic strain, see Cucumber mosaic virus (str.).
Lily yellow-flat virus, see Lily rosette virus.
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Locust witches' broom [virus], see *Robinia* brooming virus.
Loganberry dwarf virus, see Blackberry dwarf virus.
Lucerne dwarf virus, 525.
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Lycopersicum virus 2, see Tobacco mosaic virus (str.).
Lycopersicum virus 3, see Tomato spotted wilt virus.
Lycopersicum virus 4, see Tomato bushy stunt virus.
Lycopersicum virus 5, see Tomato big bud virus.
Lycopersicum virus 6, see Tomato bunchy top virus.

Maize and sorghum mosaic virus, see Sugar-cane mosaic virus (str.).
Maize mosaic virus, 525.
Maize mottle virus, see Maize streak virus (str.).
Maize streak virus, 526.
Maize streak virus, cane-streak strain, see Maize streak virus (str.).
Maize streak virus, mottle strain, see Maize streak virus (str.).
Maize streak virus, typical strain, see Maize streak virus.
Maize stripe disease virus, 526.
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Manihot virus 1, see Cassava mosaic virus.
Manila bunchy top virus, see Abaca bunchy top virus.
Marmor abaca, see Abaca bunchy top virus.
Marmor abutilon, see Abutilon variegation virus.
Marmor agropyri, see *Agropyron* mosaic virus (str.).
Marmor agropyri var. *flavum,* see *Agropyron* mosaic virus (str.).
Marmor agropyri var. *typicum,* see *Agropyron* mosaic virus (str.).
Marmor angliae, see Potato paracrinkle virus.
Marmor annularium, see Tobacco ring spot virus.
Marmor arachidis, see Groundnut rosette virus.
Marmor astri, see Peach asteroid spot virus.
Marmor astrictum var. *aucuba,* see Cucumber green-mottle mosaic virus (str.).
Marmor astrictum var. *chlorogenum,* see Cucumber green-mottle mosaic virus.
Marmor aucuba, see Potato *aucuba* mosaic virus.
Marmor aucuba var. *canadense,* see Potato aucuba mosaic virus.
Marmor betae, see Beet mosaic virus.
Marmor brassicae, see Turnip mosaic virus.
Marmor campestre var. *galbanum,* see Wheat mosaic virus (str.).
Marmor campestre var. *typicum,* see Wheat mosaic virus (str.).
Marmor cepae, see Onion yellow dwarf virus.
Marmor cerasae, see Cherry mottle leaf virus.
Marmor constans, see Tobacco green mosaic virus.
Marmor cruciferarum, see Cauliflower mosaic virus.
Marmor cucumeris var. *commelinae,* see Cucumber mosaic virus (str.).
Marmor cucumeris var. *judicis,* see Cucumber mosaic virus (str.).
Marmor cucumeris var. *lilii,* see Cucumber mosaic virus (str.).
Marmor cucumeris var. *phaseoli,* see Cucumber mosaic virus (str.).
Marmor cucumeris var. *upsilon,* see Potato virus Y.
Marmor cucumeris var. *vignae,* see Cucumber mosaic virus (str.).
Marmor cucumeris var. *vulgare,* see Cucumber mosaic virus.
Marmor dahliae, see Dahlia mosaic virus.
Marmor dodecahedron, see Tomato bushy stunt virus.
Marmor dubium var. *annulus,* see Potato virus X.
Marmor dubium var. *obscurum,* see Potato virus X (str.).
Marmor dubium var. *vulgare,* see Potato virus X.
Marmor efficiens, see Pea mottle virus.
Marmor erodens var. *severum,* see Tobacco etch virus (str.).
Marmor erodens var. *vulgare,* see Tobacco etch virus.
Marmor euonymi, see Euonymus variegation virus.
Marmor flaccumfaciens, see Rose wilt virus.
Marmor fragariae, see Strawberry crinkle virus.
Marmor graminis, see Brome mosaic virus.
Marmor iridis, see Iris mosaic virus.
Marmor laburni, see Laburnum variegation virus.
Marmor lacerans, see Peach X-disease virus.
Marmor lactucae, see Lettuce mosaic virus.
Marmor laesiofaciens, see Southern bean mosaic virus.
Marmor laesiofaciens var. *minor,* see Southern bean mosaic virus (str.).
Marmor leguminosarum, see Pea mosaic virus.
Marmor lethale, see Tobacco necrosis virus.
Marmor lineopictum, see Peach line-pattern virosis virus.
Marmor ligustri, see Privet variegation virus.
Marmor maidis var. *mite,* see Maize streak virus (str.).
Marmor maidis var. *sacchari,* see Maize streak virus (str.).
Marmor maidis var. *typicum,* see Maize streak virus.
Marmor mali, see Apple mosaic virus.
Marmor marginans, see Strawberry yellow edge virus.
Marmor matthiolae, see Stock mosaic virus.
Marmor medicaginis, see Lucerne mosaic virus.
Marmor medicaginis var. *solani,* see Lucerne mosaic virus (str.).
Marmor medicaginis var. *typicum,* see Lucerne mosaic virus.
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Marmor oryzae, see Rice dwarf virus.
Marmor pallidolimbatus, see (Flowering) Cherry banded chlorosis virus.
Marmor passiflorae, see Passion fruit woodiness virus.
Marmor persicae, see Peach mosaic virus.
Marmor phaseoli, see Bean mosaic virus.
Marmor pisi, see Pea enation mosaic virus.
Marmor pyri, see Pear stony pit virus.
Marmor repens, see Pea wilt virus.
Marmor rosae, see Rose mosaic virus.
Marmor rubi, see Raspberry mosaic virus.
Marmor rubiginosum, see Cherry rusty mottle virus.
Marmor sacchari, see Sugar-cane mosaic virus.
Marmor santali, see Sandal leaf-curl mosaic virus.
Marmor scillearum, see Ornithogalum mosaic virus.
Marmor secretum, see Dodder latent mosaic virus.
Marmor solani, see Potato virus A.
Marmor tabaci, see Tobacco mosaic virus.
Marmor tabaci var. *artum*, see Tobacco mosaic virus.
Marmor tabaci var. *aucuba*, see Tobacco mosaic virus (str.).
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Marmor tabaci var. *deformans*, see Tobacco mosaic virus (str.).
Marmor tabaci var. *immobile*, see Tobacco mosaic virus (str.).
Marmor tabaci var. *lethale*, see Tobacco mosaic virus (str.).
Marmor tabaci var. *obscurum*, see Tobacco mosaic virus (str.).
Marmor tabaci var. *plantaginis*, see Tobacco mosaic virus.
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Marmor trifolii, see Clover vein mosaic virus.
Marmor tritici, see Wheat mosaic virus.
Marmor tritici var. *fulvum*, see Wheat mosaic virus (str.).
Marmor tritici var. *typicum*, see Wheat mosaic virus.
Marmor tulipae, see Tulip breaking virus.
Marmor umbelliferarum, see Celery mosaic virus.
Marmor vastans var. *agalliae*, see Potato yellow dwarf virus (str.).
Marmor vastans var. *lethale*, see Potato yellow dwarf virus (str.).
Marmor vastans, var. *vulgare*, see Potato yellow dwarf virus.
Marmor veneniferum, see Rose streak virus.
Marmor virgatum var. *typicum*, see Wheat mosaic virus (str.).
Marmor virgatum var. *viride*, see Wheat mosaic virus (str.).
Marmor viticola, see Vine mosaic virus.
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Medicago virus 2, see Lucerne mosaic virus.
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Medicago virus 4, see Lucerne witches' broom virus.
Michigan rosette virus, see Peach rosette mosaic virus.
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Minuor ruborum, see Raspberry decline virus.
Mistletoe virus, see Lucerne witches' broom virus.
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Mosaikkrankheit des Tabaks [virus], see Tobacco mosaic virus.
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Musivum astrictum, see Cucumber green-mottle mosaic virus.
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